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Cognitive factors maintaining persecutory delusions in psychosis the contribution of depression

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**Cognitive factors maintaining persecutory
delusions in psychosis: the contribution of
depression**

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King's College London
University of London**

2012

To Cecilia Meerovna Lifshitz

Abstract

Background

Persecutory delusions are one of the most common and distressing symptoms of psychosis. Many studies indicate an association of persecutory delusions with depression. A direct role for depression-related cognitive factors in the maintenance of persecutory delusions has not been systematically examined, despite such processes being implicated in a cognitive model.

Objectives

To determine whether depression in people with persecutory delusions is associated with the same cognitive factors implicated in major depressive disorder, and to examine these factors as predictors of the persistence of persecutory delusions over time.

Methods

A systematic literature review formed the basis of two linked studies: one cross-sectional and one longitudinal. In the first study, 60 participants with persecutory delusions and schizophrenia spectrum diagnoses were classified into two groups, according to whether or not they met ICD-10 criteria for major depression. Assessments were made of delusions, depression and key cognitive factors from the literature: schematic beliefs, avoidance, rumination, memory specificity and problem solving. The groups' scores were compared, and the same comparisons were made between 30 participants with non-psychotic depression and 30 non-clinical controls. For the second study, 54 participants with delusions were re-assessed six months later, and predictors of symptom persistence were examined.

Results

50% of participants with persecutory delusions met diagnostic criteria for major depression. With baseline paranoia levels controlled, higher baseline depression predicted higher paranoia six months later. Negative schematic beliefs about the self and problem solving deficits predicted the persistence of both paranoia and depression over time.

Conclusions

Coexisting depression predicts the persistence of persecutory delusions, suggesting a causal association. Trials are warranted of depression-related therapeutic techniques for people with delusions, including those that target negative schematic beliefs about the self. An improved understanding of the mechanisms that maintain paranoid beliefs can enable the development of better treatments.

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Chapter 1. Introduction: persecutory delusions

The present thesis concerns the processes that can affect the persistence over time of persecutory delusions. A multifactorial approach is taken, with the specific goal of moving towards a greater understanding of cognitive and affective contributing factors. Given a theoretical account that proposes that problems of affect may act as important drivers of persisting psychosis (Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002), recent research has examined the role of anxiety and related processes in delusion formation and persistence (e.g. Freeman & Garety, 1999; Startup, Freeman, & Garety, 2007). The focus of the present investigation is the role of depression. The causal mechanisms involved will be approached at a cognitive-behavioural level of explanation, so that the findings can guide developments in psychological therapy as well as refining theoretical models.

In this first chapter, the phenomenon of persecutory delusions is defined, and its clinical relevance is established. Current treatment problems are noted, and several theoretical accounts are introduced that have sought to improve the understanding of relevant causal mechanisms. A rationale is traced for examining the complex set of maintenance processes that can perpetuate this highly problematic symptom. An overview of the existing strands of relevant research underpins a shift in focus to the role of depression, which will lead the next chapter.

1.1 Definition

An individual experiencing a persecutory delusion holds the unfounded or unrealistic belief that harm is occurring, or is going to occur, to him or her, and that the persecutor has the intention to cause harm (Freeman & Garety, 2000). The precise definition of a delusion has attracted much attention and debate (Garety & Hemsley, 1994; Garety, 1985; Rodrigues & Banzato, 2010). A belief is increasingly likely to be classified as a delusion, in the sense of a clinical symptom, the more it is implausible, seemingly unfounded, strongly held, preoccupying, distressing and unshared by others (Oltmanns, 1988; Freeman, 2007). It is possible that persecutory delusions in particular are easier to diagnose and study than the other most common types - referential and grandiose delusions - because the distressing nature of the content of persecution tends to cause a high degree of preoccupation and distress, as well as help-seeking or safety-seeking behaviours that can come to the attention of mental health services.

1.2 The context of schizophrenia

Delusions of persecution can occur in the context of a number of psychiatric and neurological disorders, but the phenomenon is most commonly associated with the syndrome of schizophrenia. The term "schizophrenia" means "a splitting of the mind". It was coined by Eugen Bleuler in 1908 to emphasise what he viewed as the essence of the disorder: the breaking of the connections between cognition and emotion (Bleuler, 1950). The modern definition is pragmatic: current diagnostic systems such as the ICD-10 and DSM-IV-TR comprise operational criteria for a range of symptoms as observed or described by the patient, of which a certain number must be present for a certain period of time in order for the diagnosis to be given (World Health Organization, 1992; American Psychiatric Association, 2000).

Classification systems are based on clusters of problems, reflecting the clinical observation that psychiatric symptoms tend to co-occur. This observation may result in part from comorbidity bias: if each of a set of phenomena increases the chances of a person coming to the attention of clinical services, then the phenomena are likely to be perceived as clustered, potentially beyond their real underlying correlation (Maric et al., 2004; Bak, Drukker, Van Os, & Delespaul, 2005). No individual symptom is necessary or sufficient in itself to confer a diagnosis of schizophrenia, so people with the same diagnosis can have very different presentations: a state of affairs which has driven criticism of the construct validity of the diagnosis (Bentall, 1990). Problems of predictive validity (the diagnosis of schizophrenia does not indicate how individuals will respond to treatment, or what their prognosis might be) also contribute to doubts about the existence of a single underlying disease entity.

It has been proposed that the current approach to classifying psychoses should be overhauled in its entirety on a dimensional basis, which could capture more faithfully the observable phenomenology, and would make explicit the connections between psychotic symptoms and the salient experiences of any healthy human being (Van Os, 2009; Bentall, 2006). The diagnostic category of schizophrenia is unlikely to disappear from the clinical classification systems of the near future, because a better alternative has yet to be agreed that would have sufficient clinical utility to justify such revolution (Tandon & Maj, 2008; Möller, 2009). However, for research purposes, closer specification of participants' symptoms can increase the sensitivity and specificity of findings beyond the levels afforded by a diagnostic category design.

1.3 *Single symptom research*

Persons (1986) proposed that studying specific psychological phenomena (e.g. persecutory delusions) rather than diagnostic categories (e.g. schizophrenia) affords a number of advantages, including the avoidance of participant misclassification, the opportunity to study individual elements of experience in great detail, and the utility of findings for further theoretical development, "particularly the development of coherent, elaborate hypotheses linking clinical phenomena to underlying mechanisms" (Persons, 1986).

A single symptom approach has illuminated the richness of delusional belief systems and the mechanisms of their effects on behaviour, which had not previously been captured by studies that focused on the diagnosis of schizophrenia. Delusions are complex, multi-faceted processes (Garety & Hemsley, 1994) which can vary widely in dimensions such as content, conviction, preoccupation, distress and functional impairment (Haddock, McCarron, Tarrier, & Faragher, 1999). Garety et al. (2005) found that in a sample of 100 deluded participants, delusional conviction was associated with reasoning biases and not with depression. Other studies (e.g. Freeman & Garety, 1999; Startup et al., 2007) have shown delusional distress to be associated with affective disturbance. The different dimensions of psychotic experience have been found to respond differently to treatment: antipsychotic medication may reduce preoccupation with delusional beliefs without much impact on their conviction (Mizrahi et al., 2006).

Furthermore, different delusion sub-types according to content (e.g. persecutory or grandiose) have been associated with different information processing biases, and these belief sub-types might accordingly respond to different treatment approaches. For example, Jolley et al. (2006) found, among a mixed group of people with different delusions, that only those with a combination of both persecutory and grandiose beliefs showed a self-serving attributional style (externalising the causes of negative events). Menon et al. (in press) found that a group of people with delusions of reference did not show the hasty data gathering bias that has consistently been found in people with persecutory delusions (e.g. Corcoran et al., 2008; Startup, Freeman, & Garety, 2008). Using experience sampling methodology to conduct a fine-grained analysis of the shifting occurrence of delusion-related thoughts in a group of people with psychosis over multiple points through 7 consecutive days, Ben-zeev et al. (2011) found that hallucinations predicted subsequent thoughts relating to delusions of control and of reference, but not grandiosity. These differential associations

suggest that investigating delusion sub-types individually may give more reliable results than studying the delusion category as a whole. Research interest in delusions of persecution is driven by their high prevalence and clinical prominence.

1.4 The clinical significance of persecutory delusions

Persecutory delusions are one of the most common and distressing symptoms of psychosis. The World Health Organisation conducted an international prospective study of 1379 individuals contacting psychiatric services for the first time with symptoms of schizophrenia (Sartorius, Jablensky, & Korten, 1986). Persecutory delusions were the second most common psychotic symptom found, occurring in almost 50% of respondents (delusions of reference were the most common symptom). Stompe et al. (1999) reported that persecution was the most common delusional belief type among 126 Austrian and 108 Pakistani people diagnosed with schizophrenia. Persecutory delusions are associated with strong negative affect, and are the delusion type most likely to be acted upon (Appelbaum, Robbins, & Roth, 1999; Wessely et al., 1993). The presence of persecutory delusions has been found to predict hospital admission on first contact with psychiatric services (Castle, Phelan, Wessely, & Murray, 1994).

1.5 Treatment

Neuroleptic medication is the conventional first line of treatment for schizophrenia, and the clinical guidelines report distinct improvements in positive symptoms associated with these drugs (National Institute for Health and Clinical Excellence, 2009). However, it has been estimated that between a quarter and a half of people treated in this way continue to experience distressing hallucinations and/or delusions (Garety, Fowler, & Kuipers, 2000). One of the largest clinical trials found that 74% of schizophrenia patients chose to discontinue their medication within 18 months due to inefficacy or intolerable side effects (Lieberman et al., 2005). Furthermore, non-compliance with medication regimes of 40%-50% has been reported (Lacro, Dunn, Dolder, Leckband, & Jeste, 2002).

Cognitive Behavioural Therapy (CBT) is an effective psychological treatment for psychosis (Wykes, Steel, Everitt, & Tarrier, 2008; Zimmermann, Favrod, Trieu, & Pomini, 2005), as reflected in the guidelines published by the National Institute for Health and Clinical Excellence (NICE, 2009). The symptom improvements achieved during therapy are accompanied by correlated attenuations in brain responses to threatening stimuli (Kumari et al., 2011). CBT is usually used in combination with antipsychotic medication, but recently has shown promising results even without

pharmacotherapy (Morrison et al., 2011). However, studies showed that 30-50% of individuals with persistent positive symptoms did not measurably benefit from "first generation" CBT interventions for psychosis (Kuipers et al., 1998). The most recent meta-analysis reported that although positive symptoms show beneficial effects, results are inconsistent for specific effects on delusions (NICE, 2009). A recent literature review of the effectiveness of CBT for persecutory delusions specifically (Garety, Bentall, & Freeman, 2008) concluded that effects were of small to moderate size, and that the mechanisms of change have not yet been confirmed.

Development of better treatments requires an improved understanding of the mechanisms which maintain paranoid beliefs. Freeman (2011) argued that specific theoretical advances regarding such causal mechanisms should be followed by adequately controlled intervention studies, in which each theoretically driven factor is manipulated, and corresponding improvements in delusions can be taken to indicate a causal effect. From an "interventionist-causal" perspective (Kendler & Campbell, 2009), this is the optimal pragmatic research route to improving therapeutic interventions.

1.6 Towards a psychological understanding of persecutory delusions

"Madness is when other people choose to stop trying to understand you."
(Rufus May, 2000; quoted in Laurance, 2003)

For Jaspers (1912), the defining hallmark of delusions was an irreducible quintessential incomprehensibility. Where neurosis may be seen as an exaggeration of ordinary emotional reactivity (Roth, 1963), psychosis implies a disconnection from reality: "madness proper" (Jaspers, 1912). For most of the twentieth century, mental health professionals were taught that talking to patients about their delusions would be a waste of time, and might even be harmful (Mayer-Gross, Slater, & Roth, 1954). A biomedical, neurochemical framework has been the dominant theoretical approach to the causes and treatment of psychosis, and from this point of view psychotic symptoms such as delusions and hallucinations can be viewed as quasi-epileptic phenomena, for which perhaps the causal lesion has yet to be discovered.

"Delusions are...empty speech acts whose informational content refers neither to self nor world and are not a symbolic expression of anything."
(Berrios, 1991)

Freud (1911) proposed that persecutory delusions arose as a defence against attacks of homosexual desire, which in turn originated from conflicts with parental figures in infancy: unacceptable love towards an other of the same sex was transformed, through mechanisms of denial and projection, into hatred for the other, and then, as justification for such hatred, into hatred and persecution by the other towards the self. This once quite popular hypothesis (e.g. Nunberg, 1938; Knight, 1940), is no longer prominent in current psychological theories of psychosis, but Freud's influence can be traced in accounts that emphasise the role of traumatic early life experiences (Morrison, Frame, & Larkin, 2003), conflicted family relations (Kavanagh, 1992) and defence mechanisms (Bentall, Kinderman, & Kaney, 1994; Trower & Chadwick, 1995).

While Freud's interest in the aetiology of delusional beliefs was fundamentally academic - he believed that psychoanalysis could not effectively be conducted with schizophrenic patients because they would be unable to form therapeutic relationships with the necessary transference - the contemporary cognitive theories that form the immediate background of the present thesis were developed in tandem with psychological therapies. These models were constructed with the explicit goal of guiding effective therapeutic techniques, and over time have been refined, with the help of insights from clinical trials and the findings of experimental research (e.g. Alford & Beck, 1994; Chadwick, Birchwood, & Trower, 1996; Turkington & Siddle, 1998; Freeman, 2011).

1.7 A dimensional view: the psychosis continuum

The endeavour from the late 1980s onwards to develop cognitive behavioural therapies for psychosis led psychologists and psychiatrists to try to make sense of the beliefs and experiences of people with schizophrenia within a conceptual framework equivalent to psychological models of the beliefs and behaviour of “healthy” people and of those with neurotic conditions such as anxiety or depression. It was suggested that psychotic symptoms should be viewed on a continuum with the mental phenomena occurring in the general population, who experience various levels of sub-psychotic events like perceptual anomalies and paranoid thoughts (Jones & Watson, 1997; Chapman & Chapman, 1980; Van Os, Hanssen, Bijl, & Ravelli, 2000; Strauss, 1969). Groups of people with psychosis and with non-clinical psychotic experiences were studied comparatively so that insights could be gained into the relationship between healthy variation and clinical pathology, so that specific pathogenic factors might be identified that lead to manifestations of distress and functional impairment. It was found that certain groups of non-clinical individuals, such as members of new

religious movements, reported very similar patterns of unusual experiences and delusional ideation observed in people with psychosis; the important difference was that they experienced significantly less distress and preoccupation with their beliefs (Smith, Riley, & Peters, 2009; Day & Peters, 1999; Peters, Day, McKenna, & Orbach, 1999). Population surveys confirmed the prevalence rates of self-reported psychotic experiences as being significantly higher than those of clinical psychotic disorders (Johns et al., 2004; Freeman, 2006), showing that psychotic symptoms are not exclusive to people with diagnosable mental health problems.

1.7.1 The paranoia continuum

Based on a review of ten studies of persecutory ideation in non-clinical samples, Freeman (2007) estimated that 10-15% of the general population experience paranoid thoughts on a regular basis. In order to examine the phenomenology of this paranoia in greater detail than is feasible in epidemiological research, Freeman et al. (2010) used a virtual reality simulation which would present exactly the same environment to three sets of participants, whose reported paranoid thoughts could then be compared: a group with persecutory delusions as part of a schizophrenia spectrum disorder, a group with high non-clinical paranoia, and a non-clinical non-paranoid control group. A battery of questionnaires and tasks were also used, to assess cognitive and affective processes with hypothesised relevance. The authors found that increasing paranoia across the groups was accompanied by increasing levels of depression, anxiety, anomalous experiences and traumatic past life events. The only factor found to be specific to the clinical delusions group alone was a hasty data gathering style (“jumping to conclusions”).

1.8 Reasoning bias: jumping to conclusions

Garety and colleagues (Hemsley & Garety, 1986; Garety & Hemsley, 1994; Garety, Hemsley, & Wessely, 1991) proposed that people with delusions have a tendency to “jump to conclusions” (JTC) in their reasoning style: to accept a particular explanation for experience after seeing fewer pieces of evidence than would groups without delusions. This data gathering bias has consistently been shown in people with delusions (Blackwood, Howard, Bentall, & Murray, 2001; Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Garety & Freeman, 1999). Groups with specifically persecutory delusions have repeatedly shown the JTC bias, even those with diagnoses other than schizophrenia (Corcoran et al., 2008; Startup et al., 2008). Other delusion types may not demonstrate JTC so reliably (Menon, in press). Of course, such a reasoning bias could not be sufficient on its own to precipitate a persecutory

delusion, as hasty decision-making could not explain the unusual and characteristically threatening content of the belief.

1.9 Attribution bias and the defence of self-esteem

Bentall and colleagues (1994) proposed that persecutory delusions serve as an attempt to protect self-esteem: that externalising attributions are made for negative events (others are blamed), which decrease the likelihood of underlying negative views of the self reaching conscious awareness. Empirical studies consistently find that people with persecutory delusions are significantly more likely than controls to make external attributions, particularly personal ones (blaming other people rather than chance circumstances) for negative events (reviewed by Bentall et al., 2001; Garety & Freeman, 1999). The assertion that this attribution bias functions to defend self-esteem has been somewhat controversial; this will be discussed in the next chapter, with reference to the possible role of depression.

1.10 Anomalous experiences

“...the delusional belief is not being held “in the face of evidence normally sufficient to destroy it,” but is being held because of evidence powerful enough to support it. Where the patient may differ from a normal observer is not in the manner of drawing inference from evidence but in the kinds of perceptual experience that provide the evidence from which the inference is to be drawn.”
(Maher, 1974)

Maher’s highly influential account (1974) was based around the central premise that delusions are the result of an individual with primary perceptual anomalies (and no necessary cognitive impairment) making sense of unusual sensory experiences. Once the delusional belief system is established, it is reinforced by the anxiety reduction that results from finding an explanation for those puzzling experiences, and then persists in the same way as any other strongly held belief held by, for example, a scientist or a religious person. Maher’s proposal is consistent with findings that many people with delusions experience unusual sensory phenomena (e.g. Liddle & Barnes, 1990), that the onset of hearing impairment in members of the general population predicts later psychotic experiences (Thewissen et al., 2005), and that experimentally-induced hearing impairment by hypnosis can cause paranoia (Zimbardo, Andersen, & Kabat, 1981).

A simple one-factor model of perceptual disturbance cannot explain all of the relevant available evidence. Delusions have been reported to occur without any accompanying

perceptual anomalies (Chapman & Chapman, 1988). Furthermore, a body of evidence has been presented, which suggests systematic bias in the reasoning processes and attributional style of participant groups with delusions (Bentall et al., 2001; Garety & Freeman, 1999). However, anomalous perceptual and interoceptive experiences have remained prominent in subsequent, more complex theories of delusion formation and maintenance (Morrison, 2001; Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). One of the theoretical advantages of the “anomalous perception” factor is that it can provide a link between biological and psychological levels of explanation. It is widely accepted that neurochemical changes can affect perception, and thus the neurotransmitter abnormalities commonly reported in the biological literature about psychosis can be understood to manifest in the phenomenology of experience.

1.11 Linking levels of explanation, and the theory of salience

Advocates of a cognitive neuropsychiatric approach point out the potential for vivid insights and conceptual growth that can be achieved when findings are triangulated from different perspectives, and when paradigms from one research field are applied to another (David, 1993). It has been proposed that in order to move towards a full understanding of delusions, of *how* particular combinations of predisposing and precipitating factors might interact to cause certain experiences, we must connect social, psychological and neurobiological levels of explanation, assembling the chain from genotype to phenotype (Garety et al., 2001; Garety, Bebbington, Fowler, Freeman, & Kuipers, 2007). Several authors have drawn illuminating connections between different levels of analysis of delusions (Garety et al., 2007; Kapur, 2003; Myin-Germeys, Delespaul, & Van Os, 2005). Perhaps the most widely cited of these, Kapur’s (2003) “aberrant salience” account, proposed that dopamine in all brains mediates the assignment of stimuli with motivational salience, and that in psychosis a neurochemical dysregulation of dopamine transmission disrupts the normal process of contextually driven salience attribution, causing aberrant assignment of significance to external objects and representations. The psychological process that makes sense of puzzlingly significant experiences produces delusional beliefs, which are shaped by the individual’s own cognitive, psychodynamic and cultural context.

This account accommodates many interesting features of the empirical literature on delusions, including the phenomenon of “delusional perplexity”, which often accompanies the onset of psychosis, the wide variation in content of delusions and hallucinations, the effect of dopamine agonists on precipitating psychotic phenomena and the beneficial effect on many patients of antipsychotic drugs, which block

dopamine transmission and “provide the platform for a process of psychological resolution” by dampening the aberrant salience of stimuli, so that disturbing experiences are less compelling. Van der Gaag (2006) extended the aberrant salience account to conceptualise how pharmacotherapy and psychological therapy can work together towards resolving psychotic symptoms: once experiences of aberrant salience have been dampened with antipsychotic drugs, cognitive and behavioural techniques can address the narrative that has been constructed to account for dopamine-induced psychotic experiences.

Ultimately, the implication of Kapur's account is that stopping antipsychotic medication leads to the return of chemical dysregulation, a reinvestment of aberrant salience in dormant ideas and experiences, and a relapse of psychosis. In its simplest form, this influential and highly compelling theory cannot account for the findings that some psychotic patients experience no benefit from dopamine-blocking medications (Lieberman et al., 2005; Hellewell, 1999), that some recover without ever taking them (Bola & Mosher, 2003), and that others discontinue medication without subsequent relapse. As already noted, there is clearly a very great deal of heterogeneity in the vulnerabilities, precipitators and subsequent recovery trajectories of different people with psychosis. Even when considering a single symptom such as persecutory delusions, there are likely to be many contributing factors, and each factor might be described as “an insufficient but non-redundant part of an unnecessary but sufficient condition” for the causation of the symptom (Mackie, 1974; Freeman, 2011). No one causal factor can be invoked to explain the occurrence of the symptom in all cases.

1.12 A multifactorial approach

Garety and Freeman (1999) conducted a critical review of cognitive approaches to delusions and the evidence available to evaluate them, and found that the best supported model would be a multifactorial account encompassing biases of perception and of judgement as well as disturbances of affect. Groups of people with delusions, when compared with non-clinical controls, make more externalising personal attributions for negative events, tend to gather less evidence before making task decisions (jumping to conclusions) and have low or unstable self-esteem. However, none of these features is robust enough to be found in every person who holds a delusion, and are likely to differ according to delusion sub-type (e.g. Jolley et al., 2006; Menon, Addington, & Remington, in press). The picture is complicated further by the fact that people with delusions also typically have high levels of depression and anxiety, and that these are likely to interact with psychotic phenomena. As Max Birchwood argued (Birchwood, 1998): “What is clear is that co-

morbidity in psychosis is not merely the co-occurrence of two or more disorders but rather they feed symbiotically off one another.”

Freeman et al. (2002) built on the multifactorial framework outlined above to describe a model specific to delusions of persecution. Even with a particular focus on one sub-type of delusion, a review of the evidence suggested a number of contributing factors acting in various combinations to precipitate this symptom in different individuals. The model is a vulnerability-stress model: both vulnerability and stress can be biological, psychological and/or social, and a distinction is made in the framework between factors that can contribute to the formation of threat beliefs in the first place, and factors that can perpetuate delusions once established.

The model begins with a precipitator (e.g. a stressful life event, or drug misuse), which interacts with a pre-existing vulnerability to cause unusual experiences (e.g. experiencing internally generated thoughts as heard voices), which in turn trigger a search for meaning. The impact of these anomalous experiences might be further strengthened by the effects of pre-existing anxiety and depression (which could be exacerbated by the precipitant stressor), and by cognitive biases associated with psychosis. During the search for an explanation for puzzling experiences, underlying core beliefs about the self and the world are drawn upon, and a persecutory explanation is more likely to result if the individual already believes that they are vulnerable and/or that the world is a dangerous place (these are core beliefs also associated with depression and anxiety). The externalising attributional bias described by Bentall and colleagues (1994) would make it more likely that others are blamed for negative events, and a tendency to jump to conclusions could cause an explanation to be selected that others might have thought premature.

Freeman et al.'s (2002) account conceptualised the maintenance of persecutory beliefs in a framework that would be both empirically testable and well-suited to the refinement of psychological therapies. Figure 1.1 replicates a model diagram of the mechanisms of delusional persistence. It was proposed that delusional beliefs could be maintained, once established, by the relief that comes from having found an explanation for unsettling events (and having established that something is wrong externally, rather than inside one's own mind) and by auxiliary processes that contribute to the gathering of confirmatory evidence for the threat belief and the avoidance or discarding of potentially disconfirmatory evidence. It was argued that anxiety would play a crucial role in the maintenance of threat beliefs, mediated in part by attentional biases (gathering confirmatory evidence) and safety behaviours

(avoiding disconfirmatory evidence), as described in the anxiety literature (Clark, 1999; Salkovskis, 1991). Anxiety and persecutory delusions have a clearly significant common theme: the anticipation of threat.

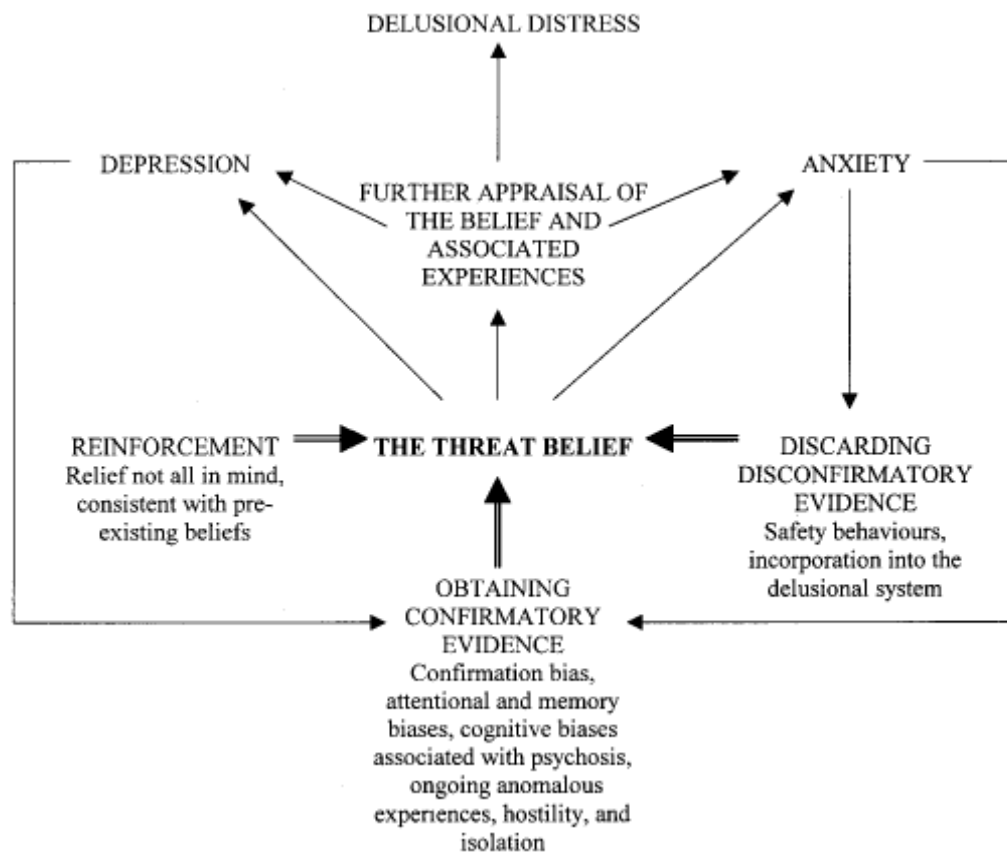


Figure 1.1. Freeman et al.'s (2002) cognitive model of the maintenance of persecutory delusions.

The prominence of anxiety in this model encouraged empirical investigation of the roles of the hypothesised mediating processes, such as worry and safety behaviours (Freeman & Garety, 1999; Freeman, Garety, & Kuipers, 2001; Freeman et al., 2007; Startup et al., 2007). The positive results that were found, particularly regarding worry predicting delusion persistence (Startup et al., 2007), led to small-scale trials of worry interventions for people with persecutory delusions (Foster, Startup, Potts, & Freeman, 2010; Hepworth, Startup, & Freeman, 2011). It was found that both worry and delusion severity could be reduced using short-term CBT, targeting worry, even without directly challenging the content of persecutory beliefs. A larger-scale, more rigorous trial is now underway, in which these findings might be extended.

1.12.1 The role of depression in a multifactorial account of the persistence of persecutory delusions

Alongside anxiety, and cognitive biases associated with psychosis, such as JTC, Freeman and colleagues' model of delusion maintenance also implicated depression. Depression was proposed to contribute to the gathering of biased evidence to support the problematic threat belief, by means of information processing biases (e.g. memory bias) and behavioural patterns (e.g. withdrawal, rumination) that can contribute to isolation and to selective engagement with stimuli. Depression is also closely associated with negative beliefs about the self, which are argued to contribute to an original vulnerability to formation of persecutory delusions.

The association between persecutory delusions and depression is likely to be bidirectional. Both the content and the wider meaning of the psychotic experience (not only am I under threat from my persecutors, but everyone else thinks that I have lost my mind) can cause or worsen depressive states (Birchwood, Iqbal, Chadwick, & Trower, 2000; Birchwood, Iqbal, Jackson, & Hardy, 2004; Birchwood, Iqbal, & Upthegrove, 2005).

Depression is known to be highly prevalent in people with psychosis (Buckley, Miller, Lehrer, & Castle, 2009), but the mechanisms of any effect on delusion persistence are unknown. The present thesis is concerned with testing this causal pathway, and examining in more detail the cognitive processes that might mediate it.

1.13 Summary

This first chapter presented a rationale for detailed investigation of the processes that contribute to the persistence of persecutory delusions over time. An overview of relevant empirical findings and theoretical models underpins a proposal that the role of depression deserves further attention.

This approach is orientated towards the refinement of cognitive behavioural therapies, and the active processes have been conceptualised at a corresponding cognitive-behavioural level of explanation. Nevertheless, maintaining the ultimate goal of an integrated bio-psycho-social understanding, it will be instructive to keep in mind the compatibility of such models with findings obtained in other fields.

In the second chapter, the evidence for a connection between depression and the positive symptoms of psychosis will be reviewed, as relatively few existing papers have reported on persecutory delusions specifically. The case will be examined for an

effect of depressive processes on positive symptom persistence, and some attention will be given to specific reports concerning delusions and paranoia. The third chapter will introduce specific cognitive mechanisms that could mediate such an effect. New empirical work testing for the hypothesised effects will then be reported, and the implications of its results will be integrated with the surrounding body of work.

In line with a multifactorial approach, it is not proposed here that depression is important in all cases of persecutory delusions, nor that its relationship to psychosis is consistent across all individuals. However, it will be seen that attention is justified to its causal and prognostic relevance in this context.

Chapter 2. Depression and positive symptom persistence

In this chapter, the case is examined for researching in detail the mechanisms connecting depression with the persistence of psychotic symptoms. A rationale for supposing the existence of such a relationship is based on a summary of prevalence data and reported associations with outcome. Three contemporary theoretical approaches are brought to bear on the relationship between depression and positive symptoms. It is seen that the causal involvement of depression would have significant clinical implications, and specific hypotheses are generated for examining key questions. A systematic review of the literature is then reported that evaluates the existing evidence for such an effect.

2.1 Introduction

Depression is very common among people with psychosis. Prevalence figures are usually found between 25% - 75%, varying with participant sample and diagnostic criteria used (Birchwood et al., 2005), and averaging around 50% (Buckley et al., 2009). The aetiological status of depression in this setting has attracted attention and prompted several thorough and comprehensive reviews (Buckley et al., 2009; Siris & Bench, 2003; Hausmann & Fleischhacker, 2002) because it has important implications for both theory and treatment. Most twentieth century accounts saw depressive symptoms occurring in people with schizophrenia either as (1) part of the core disease process in some patients, who might have schizoaffective disorder, (2) side effects of antipsychotic medication or (3) demoralisation secondary to the experience of psychotic illness. Having explored the prevalence of depression in schizophrenia spectrum disorders, the relevant risk factors and various possible causes, all of the reviews agree on the negative prognostic significance of co-occurring depression in this group. However, the causal pathways of this effect are not understood.

As noted by Hausmann & Fleischhacker (2002), in line with a multifactorial approach, a depressive syndrome in the setting of psychosis can have many different causes, and its significance for treatment will not be the same in all cases. A focus on the aetiology of depression in this group may have diverted attention away from the potential significance of the depression's consequences. Given the poor prognosis data (discussed in more detail below), combined with what is known of the effects depression can have in prolonging the symptoms of those with a range of other physical and mental health problems (Clarke, 1998), it seems likely that at least for

some people with psychosis, co-occurring depression could impede recovery by prolonging or exacerbating other symptoms. In the endeavour to improve existing treatment approaches to help people with psychosis, who often experience high levels of chronicity and impairment despite the recommended clinical care (Garety, Fowler, & Kuipers, 2000), it is important to find and to target the maintenance mechanisms that prevent symptoms from resolving.

With the development of cognitive behavioural therapies for, and of cognitive psychological models of psychosis (e.g. Garety et al., 2001) came a reconsideration of the role of emotional processes in these patients' conditions. Analyses were undertaken in more detail of the factors that might precipitate or perpetuate particular symptoms. The factors that might cause depression to emerge in somebody with psychosis were examined from a psychological point of view. Birchwood, Iqbal and Upthegrove (2005) showed that appraisal of one's psychotic experience as embodying greater loss, shame and/or entrapment is associated with higher levels of depression. Thus depression can be a natural psychological response to the content and meaning of the psychotic experience. But what of the consequences of this depression?

2.1.1 Prognostic significance

Depression occurring in people with psychosis is associated with poor functional outcomes, as agreed by existing reviews (Buckley et al., 2009; Siris & Bench, 2003; Hausmann & Fleischhacker, 2002). Among people with psychosis, those with concurrent depression respond less well to neuroleptic treatment (Gasquet et al., 2005; Himmelhoch, Fuchs, May, Symons, & Neil, 1981; Lerner, Mintzer, & Schestatzky, 1988) and experience more relapses (Birchwood, Mason, MacMillan, & Healy, 1993; Mandel, Severe, & Schooler, 1982; Roy, Thompson, & Kennedy, 1983) and more functional impairment (Birchwood et al., 1993; Sands & Harrow, 1999; Conley, Ascher-Svanum, Zhu, Faries, & Kinon, 2007) than those who are not depressed.

A handful of studies have reported a good prognosis for schizophrenia with depressive features (Emsley, Oosthuizen, Joubert, Roberts, & Stein, 1999; Mauri, Paletta, Moliterno, Colasanti, & Altamura, 2010; Taylor & Abrams, 1975; Vaillant, 1964). However, these are in the minority, and methodological shortcomings compromise the robustness of the findings (none used a comprehensive measure of depression, and the severity of psychosis in the groups that they compared was not taken into account). It is likely that a lot of patients in the older studies' groups with

“good prognosis schizophrenia” would with modern diagnostic systems be classified as having bipolar affective disorder (Taylor & Abrams, 1975).

Depression has been linked to multiple domains of poor outcome in schizophrenia spectrum disorders, but there is no established causal pathway for these effects: the mechanisms by which emotional disturbance can influence negative outcomes are not understood. One feasible role for depression in affecting outcome is that it might perpetuate positive psychotic symptoms. This possibility has not been systematically investigated, despite being implicated in cognitive models (Freeman et al., 2002; Garety et al., 2001).

2.1.2 Depression and positive symptoms

Numerous cross-sectional studies in groups with psychosis have found significant associations between higher levels of depression and more severe positive symptoms (e.g. Baynes et al., 2000; Bentall et al., 2009; Kohler, Gur, Swanson, Petty, & Gur, 1998; Lindenmayer, Grochowski, & Kay, 1991; Moller & von Zerssen, 1982).

Depression is associated with the severity of both delusions and hallucinations (Lindenmayer et al., 1991). Birchwood and Chadwick (1997) reported that hallucinatory voices were believed to be more powerful and malevolent by those who were more depressed. A belief that hallucinatory voices are omnipotent and malevolent might meet criteria for classification as a persecutory delusion.

The relationship of depression with delusions of persecution has received significant empirical attention. Freeman, Garety and Kuipers (2001) reported that 80% of a group of people with persecutory delusions showed significant levels of depression, with an overall mean BDI score of 23. Smith et al (2006) found that, among a sample of 100 recently-relapsed people with a schizophrenia spectrum diagnosis, those who were more depressed experienced persecutory delusions of greater severity and were more preoccupied and distressed by them. Bentall et al.'s (2009) large transdiagnostic study used structural equation modelling with a mixed group of people with psychosis and/or depression to show that pessimistic thinking and negative emotion had a significant association with paranoia, even when executive functioning and reasoning styles were controlled for.

2.2 Theories

The relationship between depression and positive psychotic symptoms has been conceptualised in several psychological frameworks, which differ somewhat in

emphasis. Two of the theoretical approaches introduced below are concerned specifically with delusions of persecution; the first is broader. The main assertions of each theory are, respectively, (1) that psychosis causes depression, (2) that paranoia and depression are different manifestations of a common underlying problem and (3) that depression drives (or prolongs or exacerbates) psychotic symptoms.

2.2.1 Social ranking theory and depression caused by psychosis

Max Birchwood and colleagues emphasised that the experience of having a psychotic episode can cause depression, particularly when the illness is experienced as uncontrollable (Birchwood et al., 1993) and when the experience embodies loss, shame and/or entrapment (Birchwood et al., 2000). A prospective study following 102 people with schizophrenia-spectrum disorders over 12 months found that the emergence of depression was preceded by appraisals of humiliation, loss and/or entrapment associated with illness (Iqbal, Birchwood, Chadwick, & Trower, 2000). Analysis of the patterns of depressive and psychotic symptoms over these 12 months indicated that some people had depression with onset and remission following the same trajectory as psychotic symptoms, while others developed depression later on independently of psychosis trajectory (Birchwood, Iqbal, Chadwick, & Trower, 2000). The authors concluded that post-psychotic depression developed as a result of the illness being appraised as having damaged the individual's social identity and forced them into a subordinate role without the opportunity to escape.

The evidence consistently supports a pathway to depression resulting from the experience of having schizophrenia and related disorders. However, this does not explain the experience of the significant proportion of people for whom depression precedes psychosis. Häfner and colleagues conducted thorough retrospective interviews with 232 people at admission for a first psychotic episode, and found that 83% reported clinically significant depression in the preceding period (Häfner et al., 2005). Furthermore, when the psychosis group was compared with 130 people's illness course up to first admission for an episode of major depression, the trajectory of depressive symptoms showed no difference between the two groups. The finding that 83% of first episode patients are already depressed and that depression is more common in this group than in relapsing patients (Addington, Addington, & Patten, 1998; Emsley et al., 1999) highlights the prevalence of depression that cannot be explained as a secondary consequence of psychotic breakdown. Birchwood (2003) proposed that post-psychotic depression as a psychological response is one of three core pathways to depression in people with psychosis. The second route proposed

was emotional disorder intrinsic to the psychosis itself (as described above), and the third was affective disorder caused by early developmental trauma.

2.2.2 Paranoia as a defence against negative self-evaluations

Richard Bentall's theory specifically concerns delusions of persecution, one of the most clinically prominent (Appelbaum et al., 1999; Sartorius et al., 1986; Castle et al., 1994) and best researched psychotic symptoms. He proposed that paranoia emerges as a form of camouflaged depression: a defence against negative emotional states or against low self esteem (Bentall et al., 1994; Bentall et al., 2001). The mechanism of the defence involves an externalising attributional bias for negative events: others are blamed rather than the self when bad things happen. This prevents underlying negative self-evaluations from entering consciousness.

An externalising attributional bias in paranoia has been supported empirically: people with persecutory delusions tend to attribute negative events to external causes, and in particular to other people rather than chance circumstances (Bentall et al., 2001; Garety & Freeman, 1999). The proposal that this attributional style serves a defensive function, however, has been controversial. A defence account would be consistent with relatively high or normal levels of self esteem in paranoid patients, whereas low self esteem has frequently been reported, and the discrepancies between overt and covert self-concept that a defence theory would predict have not consistently been found (Garety & Freeman, 1999). It is possible that self esteem is particularly variable in paranoid individuals (Bentall et al., 2001).

Trower and Chadwick (1995) put forward a distinction between two types of paranoia: a "Poor Me" defensive type, which rejects the perceived persecution as unfair and preserves self esteem, and a "Bad Me" low self esteem type, in which the mistreatment is seen as deserved. This deservedness dichotomy has been suggested as a helpful narrative distinction for clinical case conceptualisation and therapeutic work (Chadwick et al., 1996), but recent empirical findings are more consistent with a continuum of deservedness than with a categorical model (Bentall et al., 2008). In addition, each individual can fluctuate between the two paranoia types over time (Melo, Taylor, & Bentall, 2006).

Self esteem may not be the most useful cognitive construct for understanding the link between depression and persecutory delusions (Freeman, 2007). Drake et al (2004) found that significant associations between depression and paranoia in a first episode psychosis sample would not fit a statistical model of mediation by self esteem. Other

investigators have emphasised, instead of the balance of self esteem, specific negative beliefs about the self and about others, such as “I am vulnerable” or “others are hostile” as strongly connected to paranoia (Fowler et al., 2006). Dysfunctional core beliefs like these have long been associated with depression (Beck, 1976), and it has been proposed that holding beliefs of these kinds might predispose the individual greatly both to low mood and to paranoid thoughts.

2.2.3 Depression directly influences psychotic symptoms

Philippa Garety, Daniel Freeman and colleagues argued, as part of a multifactorial model, that the content of psychotic symptoms can be driven directly by emotional concerns (Freeman et al., 2002; Freeman & Garety, 2003; Garety et al., 2001), and furthermore that the biases of attention and processing that accompany emotional disorder can contribute (along with continuing abnormal experiences, behavioural avoidance, and a hasty data gathering style characterised by a tendency to jump to conclusions) to delusion persistence.

Compelling evidence for the causal role of depression in the development of psychotic symptoms comes from studies of transition to psychosis in high risk groups. High levels of depression in those with subclinical psychotic experiences predict subsequent precipitation of clinical psychotic symptoms (Yung, Phillips, Yuen, & McGorry, 2004; Escher, Romme, Buiks, Delespaul, & Van Os, 2002). The NEMESIS project, a three-year prospective study of a Dutch population sample of 7067 people, found that 34 out of 79 people with any psychotic symptoms at baseline assessment were also depressed (Hanssen, Bak, Bijl, Vollebergh, & van Os, 2005). These people were significantly more likely to have developed clinical psychosis by the two-year follow-up assessment than those with psychotic symptoms and no depression. Krabbendam et al. (2005) reported on the 4,670 people with three-year follow-up data. Here, given the presence of hallucinatory experiences at baseline, the likelihood of developing clinical psychosis by three-year follow-up was 28.1% for those with depressed mood at baseline, and just 11.1% for those without. It is possible that the relationship between depression and positive symptoms is specifically pronounced at this early stage of the disorder (Addington et al., 1998; Emsley et al., 1999). Depression is thought to be more prevalent in first-episode samples than in relapsing groups (Lancon, Auquier, Reine, Bernard, & Addington, 2001), and may have different interactions with other symptoms in these different phases of illness.

Daniel Freeman's research has been concerned chiefly with delusions of persecution, and particularly with the role that anxiety plays in their development. A novel

technique was devised to identify paranoid responses in the laboratory, so that attempts at manipulating these could be made (Freeman, 2008). Participants with either low levels of paranoia, high levels of subclinical paranoia or clinical delusions of persecution each entered an identical virtual reality environment populated by avatars that were programmed to act neutrally, and each participant was asked afterwards about paranoid thoughts regarding the virtual characters' intentions. Most participants interpreted the characters' behaviour as neutral or friendly, but a significant minority found them malevolent or threatening (Freeman et al., 2008). Examining paranoia across the whole continuum of its occurrence like this, rather than recruiting only patient samples, can inform inferences of causality, because the secondary consequences of psychiatric illness have not confounded the experience of subclinical participants. These studies were looking primarily for associations of paranoia with anxiety, but also found significant effects of depression. High levels of depression in participants predicted paranoid responses to neutral environments (Freeman et al., 2008). Furthermore, when groups with no paranoia, sub-clinically high paranoia and clinical delusions were compared, depression scores rose with paranoia across the three participant groups in a dose response manner (Freeman, Pugh, Vorontsova, Antley, & Slater, 2010). Those who were more depressed reported more paranoid thoughts, at all levels of severity and independently of clinical status.

2.3 A critical summary of the three theoretical perspectives

The three approaches summarised above are not mutually contradictory, and might best be thought of as each focusing on a subset of a large system of interlocking effects which gives rise to the complex and heterogeneous patterns of experience that characterise psychosis. Each theory might be most relevant to a particular subgroup of patients, to a particular stage of the illness and to a particular therapeutic strategy. Each of these three models has empirical support, with some limitations, and each can contribute its part to an understanding of the complex relationship between depression and psychosis. Each conceptualisation of the causal pathways involved carries its own set of treatment implications. These have guided therapy trials, the results of which in turn can cast additional light on the utility of the conceptual models.

Max Birchwood's research group and others have marshalled substantial evidence to prove that depression can occur as a psychological response to the experience of having a psychotic episode, when the experience is appraised in certain ways. This model does not cover depression which precedes psychosis, and does not account for associations between depression and psychosis-like experiences at a subclinical level. Richard Bentall's theory has attracted attention to the important common issues

that can be relevant in the genesis of both depression and paranoia, but the “delusions as defence” account has been undermined by inconsistent findings regarding self esteem in paranoid individuals. Daniel Freeman and colleagues’ model does make sense of these empirical findings unaccounted for by the other two theories, and achieves a high degree of explanatory flexibility by invoking a multifactorial approach. No one factor is claimed to have universal relevance for all individuals with delusions, thus the proposal that depression is a causal factor is not incompatible with the fact that some people with persecutory delusions are not depressed. The complexity of the model provides several avenues for process research, and some of these are already under investigation. The causal role of depression in this model is feasible, has not been systematically researched, and would have important clinical implications.

2.4 The rationale for a systematic review of the empirical literature

The possibility that depression plays a causal role in prolonging the positive symptoms of psychosis opens up a promising opportunity. Targeted interventions for the causal processes maintaining depression have already been developed, and show promising results in non-psychotic patients (Bell & D’Zurilla, 2009; Watkins et al., 2007; Raes, Williams, & Hermans, 2009). The use of these approaches could be indicated for people with psychosis too, and could facilitate reductions in positive symptoms as well as in mood disturbance. However, the causal relevance of depression in psychotic symptom persistence remains unconfirmed, and a systematic review of the literature is conducted here to address this. It is the aim of this review to examine the evidence linking depression with the persistence of positive psychotic symptoms.

2.5 Hypotheses

If depressive processes contribute to the persistence of positive psychotic symptoms, it is hypothesised that:

1. Among people with positive psychotic symptoms, those who are concurrently depressed will show less improvement in positive symptoms over time than those who are not depressed.
2. Effective treatment of depression in people with positive psychotic symptoms will lead to a reduction in positive symptoms as well as in depression.

2.6 Methods

2.6.1 Inclusion criteria

Studies were sought with more than one observation point of people experiencing schizophrenia spectrum disorders (schizophrenia, schizoaffective disorder or delusional disorder) that reported change over time in positive symptom severity and also included a measure of depression.

2.6.2 Procedure

A search was conducted of the SciVerse Scopus database (which includes the complete Medline database), for articles published in English before June 2011 whose title, abstract or keywords contained (schizophren* OR schizoaffect* OR psychosis OR psychotic OR delusion*) AND (depression OR depressed) AND (longitudinal OR prospective OR follow*up OR trial). Studies not meeting the above criteria were excluded. Reference lists of selected papers were inspected, and further articles meeting criteria were included in the database.

The studies reviewed comprise observational studies and treatment trials. Among treatment trials, only those were included where depression was reduced successfully, so that the evidence could be examined for parallel changes in positive psychotic symptoms.

2.7 Results

The initial search returned 7,570 results. After exclusion of irrelevant subject areas and types of publication, 3,226 citations were exported into a bibliographic software package and keyword searches were used to exclude further irrelevant material (keywords such as epilepsy, autism, dementia and others pertaining to studies obviously not relevant to this review). The remaining 294 papers were examined by hand to determine their match for the inclusion criteria. 46 articles were found to meet the search criteria. Of these, 19 were observational studies and 27 treatment trials: 12 trials of pharmacological therapy and 15 trials of psychological therapy. The studies are summarised in tables 2.1 – 2.3.

2.7.1 Methodological characteristics

The methodological quality of the studies was judged on the basis of the sample sizes and inclusion criteria used, the instruments used to measure depression and positive symptoms, the length of the follow-up period and the reported drop-out rates. For

treatment trials, it was also of interest whether or not a control group was used and whether or not group assignment was randomised.

The quality of the studies was varied, but on the whole adequate. Most of the observational studies had more than 100 participants, while most treatment trials had fewer than 100. Follow up periods used in observational studies ranged from eight weeks (Nakaya, Ohmori, Komahashi, & Suwa, 1997) up to five years (Koreen et al., 1993). The exception to this was an experience sampling study (Thewissen et al., 2011) that required participants to rate feelings of depression and paranoia ten times a day for six consecutive days. Reported drop out rates ranged from 3% to 49%.

All of the medication trials lasted 12 weeks or less, with the exception of Siris et al.'s (1994) maintenance study, which followed participants for up to a year, or until a relapse occurred. Of the 12 pharmacotherapy trials, eight used a randomised controlled design. Reported drop-out rates ranged from 0% to 50%.

The shortest follow-up period used among the psychological therapy trials was 12 weeks (Myers, Startup, & Freeman, 2011), and the longest was 24 months (Garety et al., 2008). Of the 14 psychological treatment trials, nine used a randomised controlled design. All drop-out rates from therapy were below 30%, although two studies had lost more than this proportion of participants by the final follow-up assessment (Addington et al., 2011; Peters et al., 2010).

Most studies used a widely recognised specific measure of depression: the clinician-rated Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967), or the self-report Beck Depression Inventory BDI (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Beck, Steer, & Brown, 1996), Montgomery-Asperg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979) or Calgary Depression Scale for Schizophrenia (CDSS; Addington, Addington, & Maticka-Tyndale, 1993). Some used instead a depression factor extracted from the Positive And Negative Syndrome Scale for schizophrenia (PANSS; Kay, Fiszbein, & Opler, 1987), and a few less widespread instruments. No systematic differences were evident between patterns of results obtained with different depression instruments.

Positive symptoms were measured using the PANSS positive symptom scale in most studies. A significant number - mostly pharmacotherapy trials - used instead the positive symptom scale of the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), from which the PANSS is derived. Four studies chose the Scale for

the Assessment of Positive Symptoms (SAPS; Andreasen, 1984), and five used the Psychotic SYmptom RATING Scales (PSYRATS; Haddock, McCarron, Tarrier, & Faragher, 1999), which comprise subscales relating specifically to hallucinations and to delusions. Here, as with depression measurements, no systematic differences were evident between the findings obtained by studies using different assessment measures.

Syndromes and symptoms	Trial design	
Schz = Schizophrenia	NC = Not Controlled	
SA = Schizoaffective disorder	RCT = Randomised Controlled Trial	
SF = Schizophreniform disorder	Treatment condition	
FE = First Episode		
Dep = Depression		
+ve sympts = Positive symptoms		
Neg sympts = Negative symptoms		
Sex		
F = Female	SE = Self Esteem	
M = Male	TAU = Treatment As Usual	

Measures

BCSS =	Brief Core Schema Scales (Fowler et al., 2006)
BDI =	Beck Depression Inventory (Beck et al., 1961; 1996)
BPRS =	Brief Psychiatric Rating Scale (Overall & Gorham, 1962)
CDSS =	Calgary Depression Scale for Schizophrenia (Addington et al., 1993)
CGI =	Clinical Global Impression scales (Guy, 1976)
CPRS =	Comprehensive Psychopathological Rating Scale (Åsberg, Montgomery, Perris, Schalling, & Sedvall, 1978)
DASS =	Depression Anxiety Stress Scales (Lovibond & Lovibond, 1995)
GPTS =	Green et al. Paranoid Thoughts Scales (Green et al., 2006)
HRSD =	Hamilton Rating Scale for Depression (Hamilton, 1967)
IRAOS =	Interview for the Retrospective Assessment of the Onset of Schizophrenia (Häfner et al., 1992)
MADRS =	Montgomery-Asperg Depression Rating Scale (Montgomery & Asberg, 1979)
MSCS =	Montgomery Schizophrenia Change Scale (Montgomery, Taylor, & Montgomery, 1978)
PANSS =	Positive And Negative Syndrome Scale (Kay, Fiszbein, & Opler, 1987)
PAS =	Psychiatric Assessment Scale (Krawiecka, Goldberg, & Vaughan, 1977)
PDI =	Peters Delusions Inventory (Peters, Joseph, & Garety, 1999)
PSE =	Present State Examination (Wing, Cooper, & Sartorius, 1974)
PSYRATS =	Psychotic Symptom Rating Scales (Haddock et al., 1999)
SADS =	The Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978)
SANS =	Scale for the Assessment of Negative Symptoms (Andreasen, 1983)
SAPS =	Scale for the Assessment of Positive Symptoms (Andreasen, 1984)
SCID =	Structured Clinical Interview for DSM (Spitzer, Williams, Gibbon, & First, 1992)
SIPS =	Structured Interview for Prodromal Syndromes (Miller et al., 2002)

Figure 2.1. Key to abbreviations used in Tables 2.1-2.3.

Study	Participants	Dropout	Measures
Positive association			
Bergstein, Weizman and Solomon (2008)	48 Schz with delusions	35%	BDI; PDI
Crumlish et al. (2005)	101 Schz & SF FE	31%	PANSS: Dep & +ve
Dagenhardt et al. (2007)	101 Schz spectrum	31%	CDSS; BPRS
Drake et al. (2004)	257 Schz spectrum FE	28%	PANSS & PSYRATS: Dep & paranoia factors
Edwards et al. (1998)	158 Schz spectrum FE; 69 other psychosis FE	35%	BDI; BPRS
Fowler et al. (2011)	301 Schz spectrum with +ve symps	18%	BDI; BCSS; SAPS; PANSS
Häfner et al. (2005)	232 Schz FE	22%	PSE, SANS, IRAOS
Koreen et al. (1993)	70 Schz FE	24%	SADS affective and psychotic scales
Lancon et al. (2001)	27 Schz spectrum	33%	CDSS; PANSS
Nakaya et al. (1997)	89 Schz	3%	HRSD; PANSS
Norman & Malla (1994)	55 Schz	5%	BDI; SAPS
Rooke & Birchwood (1998)	49 Schz	4%	BDI; PAS
Siegel et al. (2006)	98 Schz (inc 45 FE)	6%	HRSD; SAPS
Tarrier et al. (2001)	87 Schz with +ve symps	17%	BDI; BPRS; SANS; PAS
Thewissen et al. (2011)	82 Schiz spectrum; 39 high schizotypy; 37 non-clinical	14%	ESM: ten times per day rate Dep & paranoia
No association			
Aguilar et al. (1997)	96 Schz spectrum FE	11%	HRSD; SAPS
Carroll et al. (1999)	100 Schz	18%	MADRS; PANSS +
Lindenmayer & Kay (1987); Kay & Lindenmayer (1987)	37 young Schz	49%	PANSS / BPRS D factor; +ve subscale
Oosthuizen et al. (2002)	80 Schz or SF, FE	28%	PANSS D factor; +ve subscale
Schennach-Wolff et al. (2011)	224 Schz	34%	HRSD; PANSS +ve

Table 2.1. Observational studies in functional psychosis.

Study	Duration	Results
Positive association		
Bergstein, Weizman and Solomon (2008)	12 mo	Dep associated with delusions within each of 3 time points. Initial Dep correlates with delusions a year later.
Crumlish et al. (2005)	4 yr	Dep associated with +ve sympts at both times. No longitudinal analysis.
Dagenhardt et al. (2007)	10 mo	Dep score at a given point predicts later +ve symptom score.
Drake et al. (2004)	18 mo	Dep and Paranoia factors associated at each time point and across time points, direction of effect ambiguous.
Edwards et al. (1998)	12 mo	Those with persistent +ve sympts at all three points (8.9% of Schz spectrum pts) had higher Dep at 12mo than the people without +ve sympts at all points.
Fowler et al. (2011)	12 mo	Dep factor and negative cognition factor both predict paranoia factor, cross-sectionally and over time. negative cognition mediates the effect of Dep on paranoia.
Häfner et al. (2005)	retrospective to admission	those with Dep in early course had higher +ve symptoms at admission than those who had not been depressed.
Koreen et al. (1993)	Biweekly / monthly up to 5 yr	of 808 psychotic ratings, 210 concurrently depressed. Of 1754 nonpsychotic ratings, only 70 depressed. Co-occurrence statistically significant. Dep & +ve sympts correlated within patients. No cross-lagged analyses.
Lancon et al. (2001)	admission; 6 mo after discharge	Dep and +ve sympts both decreased. Dep & +ve correlated at admission; no correlation at follow-up.
Nakaya et al. (1997)	9 wk	Dep and +ve symptom declines are correlated; no correlation at baseline.
Norman & Malla (1994)	12 - 29 mo	Dep and +ve sympts consistently correlated for most patients.
Rooke & Birchwood (1998)	30 mo	At follow-up Dep associated with delusions, and trend with hallucinations. No longitudinal analysis.
Siegel et al. (2006)	up to 8 yr	+ve sympts at intake predict Dep at follow-up (not the other way around).
Tarrier et al. (2001)	3 mo	Dep change correlated with +ve symptom change.
Thewissen et al. (2011)	ESM 10x day 6 days	Dep associated with paranoia; initial Dep predicts longer duration of paranoia.
No association		
Aguilar et al. (1997)	12 mo	Dep & +ve symptoms uncorrelated at baseline, associations over time not reported. Hopelessness predicts poor functional outcome.
Carroll et al. (1999)	12 wk	Dep & +ve symptoms uncorrelated at baseline; change scores also uncorrelated.
Lindenmayer & Kay (1987); Kay & Lindenmayer (1987)	2 yr	nonsignificant trend towards a correlation between Dep at intake and more +ve symptom improvement 2yrs on.
Oosthuizen et al. (2002)	6 mo	Dep consistently assoc Neg sympts, not +ve.
Schennach-Wolff et al. (2011)	biweekly admission to discharge	Dep consistently assoc Neg sympts, not +ve.

Table 2.1 continued. Observational studies in functional psychosis.

2.7.2 Observational Studies

One of the studies included here relates to a treatment trial where no between-group treatment effects on depression were observed, and the data are presented observationally (Tarrier et al., 2001).

Of the 20 studies listed in Table 2.1, 15 found an association between higher depression and more positive symptoms. Most consistently, relationships were found between measurements taken at the same time point, but ten of the 20 studies went on to examine whether depression at a given time point might predict the severity of subsequent positive symptoms. Six of these ten reported that depression scores at a given point in time were related to more severe positive symptoms at a later point (though one of these studies assessed symptoms retrospectively rather than prospectively). Four studies looked for but did not find this predictive effect, although one of these four did find the opposite: that initial positive symptom levels predicted subsequent depression (Siegel et al., 2006). Ten studies did not examine the predictive effect of depression on later positive symptoms. However, two of these ten did find that reductions in depression scores and positive symptoms over time were correlated with each other (Nakaya et al., 1997; Tarrier et al., 2001). Another selected a group of participants with persistent positive symptoms across all three of the study's observation points, and found that this group were more depressed than those whose positive symptoms were less persistent (Edwards, Maude, McGorry, Harrigan, & Cocks, 1998).

Among eight observational studies of first episode samples reviewed here, five found a relationship between higher depression scores and more severe positive symptoms: two found that depression levels predicted positive symptoms longitudinally; one found that those with the most persistent positive symptoms were most depressed; the other two did not look for a longitudinal relationship, but found cross-sectional associations.

Notably, all four studies that measured persecutory or delusional ideation specifically found that depression at a given time point was related to more severe subsequent paranoia (Drake et al., 2004; Fowler et al., 2011; Thewissen et al., 2011) or delusions (Bergstein, Weizman, & Solomon, 2008).

Two studies stood out as being particularly convincing (Drake et al., 2004; Fowler et al., 2011). These had the largest sample sizes of all in Table 2.1, included only people with schizophrenia spectrum diagnoses and used a prospective design with structural

equation modelling to disentangle the causal effects that might be at play between symptom dimensions.

Drake et al. (2004) assessed 257 patients with a first psychotic episode up to four times over 18 months using the PANSS and the PSYRATS, which were used to derive a depression factor score and a paranoia factor score for each assessment point. They found that higher paranoia was associated with more depression at each time point. Cross-lagged associations were weak, but models with a causal pathway between depression and paranoia fit the pattern of data significantly better than those without. The direction of the effect between depression and paranoia was ambiguous.

Fowler et al. (2011) used a similar modelling technique with a larger sample ($N = 301$) and more sensitive measures of depressive affect and cognition (the BDI-II and Brief Core Schema Scales) to compare models with either depression driving subsequent paranoia or vice versa. Cross-lagged effects were significant, and the most plausible models were those with pathways from depressive affect and cognition to paranoia, not the other way around.

Study	Participants	Dropout	Measures	Duration
Yes parallel improvement +ve symptoms				
Hogarty et al. (1995)	38 Schz / SA + Dep	13%	BDI; BPRS	12 wk
Goff et al. (1990)	14 Schz	21%	HRSD; BPRS	6 wk
Mazeh et al. (2004)	N = 19 Schz + Dep	36%	HRSD; PANSS	6 wk
Siris et al. (1994)	24 Schz / SA + Dep or Neg sympts: had benefitted from imipramine in Siris et al. (1987)	none	CGI; SADS	to relapse <1yr
No parallel improvement +ve symptoms				
Siris, Morgan & Fagerstrom (1987)	5 Schz & 28 SA, all with HRSD > 12	6%	SADS: Dep items; +ve items	6 wk
Prusoff et al. (1979)	40 Schz + Dep	50%	HRSD; BPRS	6 wk
Singh, Saxena & Nelson (1978)	60 Schz + Dep	unknown	HRSD; BPRS	6 wk
Bodkin et al. (1996)	21 Schz / SA + Neg sympts	19%	HRSD; BPRS	6 wk
Englisch et al. (2009)	20 Schz spectrum, no +ve sympts, mild Dep	5%	CDSS; HRSD; PANSS	6 wk
Zisook et al. (2009)	198 Schz / SA + subsyndromal Dep	5%	CDSS; HRSD; PANSS	12 wk
Kasckow et al. (2001)	19 Schz + Dep	11%	HRSD; PANSS	10 wk
Spina et al. (2004)	34 Schz + Neg sympts, no Dep	12%	HRSD; BPRS; SAPS	12 wk

Table 2.2. Antidepressant pharmacotherapy trials.

2.7.3 Treatment trials: medication

Table 2.2 summarises the methods and findings of 12 antidepressant medication trials. These include 11 trials that reported adding an antidepressant to participants' existing antipsychotic treatment regimens and finding subsequent reductions in depressive symptoms. Three of these 11 also reported a parallel reduction in positive psychotic symptoms. These were no more rigorously conducted or highly powered on the whole than the eight trials that found no decrease in positive symptoms. If anything, the studies that found changes in positive symptoms used smaller sample sizes.

Study	Design	Medication	Results
Yes parallel improvement +ve symptoms			
Hogarty et al. (1995)	RCT	desipramine	F only: BDI & BPRS both improved.
Goff et al. (1990)	NC	fluoxetine	improved Dep & BPRS
Mazeh et al. (2004)	NC	venlafaxine	14 patients significant improvement HRSD; in most a parallel PANSS decrease
Siris et al. (1994)	RCT	maintain imipramine or taper to placebo	those tapered down to placebo had more depressive & psychotic relapses
No parallel improvement +ve symptoms			
Siris, Morgan & Fagerstrom (1987)	RCT	imipramine	improved Dep; +ve no diff. (23 of 33 were non-psychotic at baseline.)
Prusoff et al. (1979)	RCT	amitriptyline	no diff HRSD total; change on one subscale at one of 4 assessment points. No diff BPRS total.
Singh, Saxena & Nelson (1978)	RCT	trazadone	improved HRSD; no diff BPRS.
Bodkin et al. (1996)	NC	selegiline	improved HRSD; no diff BPRS.
Englisch et al. (2009)	NC	duloxetine	improved CDSS & HRSD; PANSS +ve stably absent. (Those with significant +ve sympts had been excluded from the study.)
Zisook et al. (2009)	RCT	citalopram	improved Dep; +ve no diff.
Kasckow et al. (2001)	RCT	citalopram	improved Dep; +ve no diff.
Spina et al. (2004)	RCT	fluoxetine	improved Dep; +ve no diff.

Table 2.2 continued. Antidepressant pharmacotherapy trials.

One further study (Siris, Bermanzohn, Mason, & Shuwall, 1994) reported a comparison of maintenance antidepressant therapy against tapering to placebo for people whose depression had responded to imipramine in an earlier trial (Siris, Morgan, & Fagerstrom, 1987). The authors found that those who were tapered to placebo experienced more psychotic relapses, as well as more depressive relapses, than those who were maintained on antidepressant medication.

Authors	Participants	Dropout %	Measures	Duration
Yes parallel improvement +ve symptoms				
Addington et al. (2011)	51 clinical high risk	25%	CDSS; SCID; SIPS	18 mo
French et al. (2007)	57 help seeking at risk	26%	PANSS: Dep factor & +ve	1 yr
Garety et al. (1994)	20 Schz / SA +ve sympts	10%	BDI; BPRS	6 mo
Hall & Tarrier (2003)	25 Schz, +ve sympts & low SE	8% 2mo, 28% 5mo	HADS; PANSS +ve	5 mo
Knight, Wykes & Hayward (2006)	21 Schz spectrum	10%	BDI; PANSS +ve	12 wk
Laithwaite et al. (2007)	15 Schz (forensic)	none	BDI; PANSS +ve; PSYRATS	6 mo
Myers, Startup & Freeman (2011)	15 Schz spectrum persistent PDs, insomnia	none	DASS; GPTS; PSYRATS	3 mo
Peters et al. (2010)	74 persistent distressing +ve symptom(s)	26% 6mo, 42% 9mo	BDI; PANSS +ve	9 mo
Sensky et al. (2000)	90 Schiz	none	MADRS; CPRS	up to 18 mo
Trower et al. (2004)	38 Schz spectrum command Hal	21%	CDSS; PANSS; PSYRATS	12 mo
Garety et al. (2008)	301 Schz spectrum +ve sympts	20%	BDI; PANSS; PSYRATS	24 mo
No parallel improvement +ve symptoms				
Laithwaite et al. (2009)	15 Schz spectrum; 3 bipolar (forensic)	5%	BDI; PANSS	4 mo
Penades et al. (2006)	40 Schz + Neg sympts, minimal +ve	18%	PANSS: Dep factor & +ve	10 mo
Turkington, Kingdon & Turner (2002)	422 Schz	16%	MADRS; CPRS; MSCS	5 mo

Table 2.3. Psychological therapy trials.

Authors	Design	Intervention	Results
Yes parallel improvement +ve symptoms			
Addington et al. (2011)	RCT	CBT vs supportive therapy	Dep and +ve sympts improved both groups, but +ve sympt improvement more rapid in CBT group.
French et al. (2007)	RCT	CBT + monitoring vs TAU + monitoring	Dep and +ve sympts correlated and reduced. In CBT group bigger effect for +ve sympts than control. Not so for Dep.
Garety et al. (1994)	wait list control	CBT vs TAU	Dep and BPRS reduced in CBT group.
Hall & Tarrier (2003)	RCT	CBT for SE	Post-treat CBT group sig more improvement in Dep and +ve sympts than control. Follow-up +ve gains maintained but Dep no longer different.
Knight, Wykes & Hayward (2006)	wait list control	anti stigma group for SE	SE increased, Dep and +ve sympts decreased during therapy period, not in wait list control period.
Laithwaite et al. (2007)	not controlled	CBT for SE	Dep and PSYRATS Delusions improved, not AH scale or +ve PANSS total.
Myers, Startup & Freeman (2011)	not controlled	CBT for insomnia	All measures decreased significantly.
Peters et al. (2010)	RCT wait list control	CBT vs TAU	BDI sig reduced at post and follow-up in combined group.. +ve sympts sig reduced only in the delayed therapy group not immediate.
Sensky et al. (2000)	RCT	CBT vs befriending	post treatment both groups improved CPRS and MADRS. 9 month followup CBT group further gains on both, befriending groups lost some (sig difference).
Trower et al. (2004)	RCT	CTCH vs TAU	Dep rose in TAU not CTCH. +ve sympts rose in TAU, dropped in CTCH: difference in Delusions not Hallucinations
Garety et al. (2008)	RCT	CBT vs FI vs TAU	CBT reduced Dep and reduced Delusional Distress in those with carers compared to TAU. No sig difference in +ve sympts total.
No parallel improvement +ve symptoms			
Laithwaite et al. (2009)	not controlled	Compassion focused therapy for SE	sig reduction Dep but not PANSS +ve. Was low to begin with (mean = 9).
Penades et al. (2006)	RCT	CBT vs CRT	CBT reduced Dep; no difference +ve scores.
Turkington, Kingdon & Turner (2002)	RCT	CBT vs TAU	Dep and overall symptoms improved in CBT vs TAU, but "schizophrenia score" no difference in rates of change. No proper measure of +ve symptoms.

Table 2.3 continued. Psychological therapy trials.

2.7.4 Treatment trials: psychological therapy

Table 2.3 summarises 14 psychological therapy trials that reported improvement of depressive symptoms in people with schizophrenia spectrum disorders and also

measured positive symptoms. Eight of the 14 trials (including five out of nine randomised controlled trials) reporting improvement in depression also found a parallel improvement in positive psychotic symptoms. Garety et al. (1994), Sensky et al. (2000) and Turkington et al. (2002) did not report changes in positive symptoms specifically, but found a treatment effect, alongside depression, on global psychotic symptoms as measured by the CPRS/BPRS. Garety et al.'s (2008) large RCT found no treatment effect on PANSS positive scale total scores, but did find that those participants who had carers showed a treatment effect of CBT on delusional distress as measured by the PSYRATS. Similarly, Laithwaite et al.'s (2007) pilot study of CBT for self esteem in forensic inpatients with schizophrenia showed that PSYRATS Delusions subscale scores reduced significantly alongside depression scores, but that PSYRATS Auditory Hallucination subscale scores and PANSS positive scale total scores did not. Two studies achieving a treatment effect on depression examined but did not find any parallel changes in PANSS positive scale scores (Laithwaite et al., 2009; Penadés et al., 2006). The participants in both of these studies had very low positive symptom scores to begin with (average PANSS Positive scale total = 9 in the Laithwaite et al. study, and 11 in the Penades et al. study, where scores can range from 7 to 49).

2.7.5 Three excluded studies with some relevance

Two reports were found that did not meet inclusion criteria for the systematic review, but referred to trials that targeted affective disturbance specifically in people with psychosis (White et al., 2011; Jackson et al., 2009), which most of the psychological therapy trials above had not set out explicitly to achieve. One further paper (Lecomte et al., 1999) reported on a trial of a self esteem therapy intervention that was similar to the four reviewed above (Hall & Tarrier, 2003; Knight, Wykes, & Hayward, 2006; Laithwaite et al., 2007; Laithwaite et al., 2009).

White et al. (2011) reported on a randomised controlled trial of Acceptance and Commitment Therapy (ACT) for emotional dysfunction following psychosis, which was published too late to be included in the above study pool. 27 people with psychosis were randomised to ACT or treatment as usual (TAU). Three months after baseline assessment, significantly more of those in the ACT group had ceased to be depressed than of those the TAU group. No differences were observed between positive symptom score changes in the two groups - neither group showed a change - although the ACT group had significantly fewer crisis contacts. However, as with Laithwaite et al.'s (2009) study and Penades et al.'s (2006) study, White et al.'s (2011)

participant group entered the trial with minimal positive symptoms (PANSS Positive scale total mean = 12).

Jackson et al. (2009) conducted a randomised controlled trial of recovery-focused cognitive therapy to target depression and post-traumatic symptoms following in the wake of a psychotic episode. 66 first-episode psychosis patients were randomised to cognitive therapy or TAU. The main therapeutic targets were trauma processing and appraisals of psychotic illness embodying shame, loss and/or entrapment. Despite a beneficial effect of the intervention on traumatic symptom scores, the authors found no improvements in depression or self esteem scores in the therapy group over TAU. No measures were reported of participants' appraisals of psychotic illness, or of positive psychotic symptoms.

Lecomte et al.'s (1999) study compared a 12-week self esteem coping module against TAU in a group of people with schizophrenia, measuring changes in coping and psychotic symptoms (but not depression, which is why the study did not meet the review criteria). The authors found significant improvements in the therapy group's positive symptom scores compared to the TAU group, specifically on items about delusions, perceptual disorganization and paranoia. This study's pattern of findings was similar to those of Hall and Tarrier's (2003), Knight et al.'s (2006) and Laithwaite et al.'s (2007) self esteem therapy trials. Of all the trials mentioned here that targeted self esteem in people with psychosis, only Laithwaite et al. (2009) failed to find an effect on positive symptom scores.

2.8 Discussion

2.8.1 Observational studies

The first hypothesis of this review was that among people with positive psychotic symptoms, those who were concurrently depressed would show less improvement in positive symptoms over time than those who were not depressed.

The results of the review indicated that high levels of depression were consistently associated with high levels of positive psychotic symptoms. As a cross-sectional pattern within one point in time, this was robust; longitudinal findings were mixed. Ten studies were found which tested specifically for a predictive effect of initial depression on the severity of positive psychotic symptoms at a later point in time. Six of these succeeded in finding such an effect, all in the predicted direction. The first hypothesis was thus supported by the majority of relevant studies: positive psychotic symptoms

tended to resolve more slowly in those with high levels of depression than in those without. The strongest findings came from studies that examined delusions or paranoia specifically.

2.8.2 Treatment trials

The second hypothesis of this review was that effective treatment of depression in people with positive psychotic symptoms would lead to a reduction in positive symptoms as well as in depression levels.

2.8.2.1 Antidepressant medication trials

The medication trials reviewed indicated that antidepressant medications added to antipsychotic regimens did not show beneficial effects on positive psychotic symptoms, at least not over periods of time up to 12 weeks. Most of the antidepressant trials achieving significant reductions in depression did not report parallel decreases in positive symptoms. The interactive dynamics of polypharmacy are beyond the scope of this discussion, but it can be noted that all of the trials reviewed were short (12 weeks or less) and most had relatively small samples, some of which were selected for an absence of positive symptoms. Many of the trials had only started to detect significant changes in depression at the end of the assessment period, and it is possible that subsequent improvements in positive symptoms might have occurred without being captured by these study protocols. The short follow-up periods used and the exclusion from some of the participant samples of those with significant positive symptoms might have limited the usefulness of the resulting data for the evaluation of the above hypothesis.

2.8.2.2 Psychological therapy trials

The reviewed psychological therapy trials indicated that effective reductions in depression by means of psychological therapy were often accompanied by reductions in positive symptoms, particularly delusions. Psychological therapy trials involved longer follow-up periods (up to two years) than pharmacological trials, and the majority found that reductions in depression achieved were paralleled with improvements in positive psychotic symptoms. Most of these had not sought to reduce depression specifically, although four did target self esteem (Hall & Tarrier, 2003; Laithwaite et al., 2009; Laithwaite et al., 2007; Knight et al., 2006). Most of the trials set out explicitly to target positive symptoms. Where such trials observed reductions in both symptom dimensions, to conclude that the depression reduction caused the positive symptom reduction would be premature, but this possibility is not out of the question. Therapies such as CBT for psychosis have evolved from those

originally developed for depression, and many techniques were carried over, including those that target beliefs about the self, interpretations of events and responses to distress. It is plausible that these therapeutic approaches could exert part of their effect on psychotic symptoms via reductions in depressive features. A good test of this hypothesis would involve treating depression specifically in people who experience it alongside psychosis. Subsequent changes in positive symptoms could then be examined for significant effects.

2.8.3 Targeting depression

Only one of the psychological therapy trials included in the review set out specifically to target depression in people with psychosis (Laithwaite et al., 2009), although two more were found that targeted depression but did not meet review criteria (Jackson et al., 2009; White et al., 2011). Each of these three studies failed to provide information crucial to the testing of the review's second hypothesis, due to methodological limitations. Laithwaite and colleagues used compassion focussed group therapy with forensic inpatients who had schizophrenia or bipolar diagnoses, and White and colleagues used ACT with a mixed group of people with psychotic disorders. Both interventions achieved significant reductions in depression compared to TAU. No effects were found on positive symptoms in either trial, but this is attributable in both cases to a floor effect. Most of the participants in these studies had no positive symptoms to begin with: average PANSS positive subscale scores were 9 and 12 respectively, where scores can range from 7 to 49.

Jackson et al.'s (2009) therapy trial sought to ameliorate depression by reducing participants' appraisals of their psychotic illness as embodying humiliation, loss or entrapment. This followed Max Birchwood's social ranking model of post-psychotic depression (Birchwood, 2003), which carried the implication that by manipulating the meaning of the psychotic experience to the individual, reducing the degree of defeat and entrapment experienced, depression and its associated negative effects could be prevented. Depression showed no benefit from this intervention compared to TAU, and this was not attributable to a floor effect: baseline CDSS scores had indicated that 67% of the participants were "significantly" depressed. No measure was reported of change in the illness appraisals that the intervention sought to target, so it is unclear whether therapy failed to change these or whether it did so without correspondingly reducing depression. The approach used by Jackson and colleagues in this study differed substantially from the techniques used in therapies recommended by NICE for people with major depressive disorder (National Institute for Health and Clinical

Excellence, 2009), which have not been comprehensively trialled in people who have depression co-occurring with functional psychosis.

2.8.4 The role of self esteem

All but one of the trials reviewed that achieved reductions in depression by targeting self esteem also reported corresponding reductions in positive psychotic symptoms (Hall & Tarrier, 2003; Laithwaite et al., 2007; Knight et al., 2006). The only trial targeting self esteem that didn't find positive symptom reductions had started with a participant group with already minimal positive symptoms, as mentioned above (Laithwaite et al., 2009). Lecomte et al.'s (1999) study, though not meeting the present review criteria as it did not report depression changes, also found that a self esteem intervention led to positive symptom reductions, particularly on items relating to delusions, paranoia and perceptual disorganization. Laithwaite et al. (2007) also looked individually at different psychotic symptoms, and found that delusions reduced significantly in the treatment group while hallucinations did not. This evidence supporting the role of self esteem in positive symptom resolution (with a particular accent on delusions and paranoia) can be interpreted as supporting, to some extent, both Bentall et al.'s (1994; 2001) and Freeman et al.'s (2002) theoretical models. Both accounts proposed that negative evaluations of the self drive persecutory delusions.

Bentall and colleagues' (1994; 2001) defence model suggested that discrepancies between overt and covert self esteem are evident in people with persecutory delusions, and that a self-serving attributional bias functions to prevent the covert negative self-evaluations from entering consciousness. The finding that therapeutically targeting self esteem can cause reductions in positive symptoms, including delusions, does not provide direct support for a defence model; it can be difficult empirically to distinguish between overt and covert self-esteem, and such a distinction was not made in any of the studies reviewed here. It seems more likely that explicit self esteem was targeted and measured in these protocols. Nevertheless, evaluative beliefs about the self have strong support as a factor affecting psychotic symptoms.

Freeman and colleagues' (2002) model, following Garety et al. (2001), proposed that core beliefs about the self and the world are drawn upon when an individual tries to find an explanation for anomalous experiences (which are caused by interactions between bio-psycho-social vulnerabilities and stressors). Negative underlying beliefs thus make it more likely that a persecutory explanation is chosen: negative self-evaluations in this framework have a direct rather than defensive effect on delusion

formation, and no discrepancies between overt and covert self-evaluations are involved. The findings of trials that targeted self esteem with resulting reductions in positive symptoms are consistent with Freeman and colleagues' proposal that delusions of persecution are perpetuated by their consistency with underlying negative core beliefs. The model goes on also to implicate both depression and anxiety in the gathering of biased evidence to support persecutory beliefs. The role of anxiety has now received attention in a series of studies supporting the roles of attention to threat (Freeman, Garety, & Phillips, 2000), worry (Startup et al., 2007; Foster et al., 2010) and the use of safety behaviours (Freeman et al., 2007) (all features linked to anxiety) as potentially helpful therapeutic targets in breaking the cycle of delusion persistence. Most of the cognitive features associated with depression have not yet been systematically investigated in people with psychosis.

2.8.5 Delusions and other positive symptoms

Depression is more consistently predictive of the persistence of delusions and paranoia than of total positive symptom scores. Mixed findings have been reported regarding the prediction of positive scale totals, but every study in the present review that measured delusions or paranoia specifically found that depression predicted the persistence of such beliefs over time, or, in the case of therapy trials, that reductions in depression were accompanied by weakening of delusions. This pattern of findings is consistent with the cross-sectional observations summarised in the introductory section of this chapter: delusional or paranoid ideation has received the most empirical attention in relation to depression, and has demonstrated a robust relationship.

Three of the reviewed psychotherapy trials examined relative changes in delusions and hallucinations specifically. Garety et al. (2008) individually measured changes in hallucination frequency and distress, delusional conviction and distress, and found that delusional distress was the only one of these four parameters to show treatment effects (along with depression) from CBT. Laithwaite et al.'s (2007) self esteem therapy trial measured symptom changes using both the Auditory Hallucinations subscale and the Delusions subscale of the PSYRATS, and found that participants' Delusions scores reduced significantly with treatment, along with depression scores, while Hallucinations scores did not. Trower et al.'s (2004) trial involved an intervention specifically targeting command hallucinations. The treatment group here showed significant reductions compared to the TAU group in depression, distress, beliefs about the power of voices and the need to comply, and actual compliance behaviours. Meanwhile, the frequency, loudness and content of the voices did not change. PANSS

positive scale total scores also reduced significantly in the treatment group: within the PANSS, delusions scores showed a significant drop, but hallucinations scores did not. These findings suggest that the kinds of interventions that successfully reduce depression in people with psychosis might have quite different effects on individual psychotic symptoms.

The major role played by depression and negative schematic beliefs in Garety et al.'s (2001) and Freeman et al.'s (2002) models is in the precipitation and maintenance of delusional beliefs. Anomalous experiences like hallucinations are proposed to result from interactions of environmental stressors with pre-existing cognitive and neurochemical vulnerabilities. Emotional states can exacerbate unusual perceptual phenomena, but the primary effect of depression and negative self-appraisals is in influencing the selection of an explanation for puzzling experiences. The possible differential associations of individual psychotic symptoms with depression have yet to be fully explored, but the findings of this review support the proposal that depression has a stronger connection with the persistence of delusions than hallucinations. Depression has, ever since Beck's influential account, been associated with certain pathologies of belief: core negative beliefs about the self and other people (e.g. "I am unlovable", "others are uncaring") and conditional assumptions based on such core beliefs (e.g. "If I try to get close to people, they will reject me"). A person with depressive core beliefs (e.g. "I am defective") might also be more susceptible to other kinds of compatible distressing beliefs (e.g. "people in the street are singling me out to harm me") than somebody whose belief system is altogether more conventional.

2.8.6 Methodological issues

The wide variability in participant sample characteristics, follow-up periods and symptom measures used in these studies might have contributed to the heterogeneity in findings. The content and psychometric properties of different scales used to measure depression are a relevant example. The HRSD had long been seen as the "gold standard" for depression research, but more recently was shown to be less accurate and less internally consistent than the BDI and the MADRS, potentially not valid for use in outpatient samples (Uher et al., 2008). The PANSS depression factor is particularly open to criticism as a measure of depressive symptoms. It is composed of four items, each scored 1-7 by the assessor: Somatic Concern, Anxiety, Guilt Feelings and Depression. Thus only half of the items relate to symptoms of depression as defined by the DSM-IV or the ICD-10, and all have equal weighting. No systematic difference was evident however between the findings of studies that used adequate measures of depression and those that used more tenuous ones. With

regard to positive symptom measures, there were stronger and more consistent findings in studies where specific measures of delusions or paranoia were used than in studies which summed positive symptom scale totals.

Individual psychotic symptoms could have quite different relationships with depression, and such differential associations have yet to be further explored. If it is the case that delusions are more closely related to depression than other positive symptoms, then the use of total summed positive symptom scores in analyses may have obscured these different relationships in some studies. The PANSS positive symptom scale, which was used by most of the studies reviewed here to measure positive symptoms, is calculated by summing scores 1-7 on each of 7 items: Delusions, Conceptual disorganization, Hallucinatory behaviour, Excitement, Grandiosity, Suspiciousness / Persecution and Hostility. The items all have equal weight. The way the instrument is summed means that qualitatively very different presentations can be numerically equivalent. For example, a manic affective presentation (elevated mood, grandiosity, flight of ideas, irritability) could produce the same score as a stereotypical paranoid schizophrenic one (hallucinations, persecutory delusions, thought disorder). One might expect that depression would have negative relationships with Excitement and with Grandiosity that could counteract a positive relationship with Suspiciousness / Persecution when scores are summed to a total. This could explain the non-significant findings of some of the studies reviewed here.

2.8.7 Limitations and indications for future research

The specific hypotheses that this review sought to examine were quite different from those that most of the reviewed studies set out to test. As a result, the nature of the data was not ideal for the present purpose. This was particularly pertinent with the antidepressant medication trials, where participant selection criteria and short follow-up periods severely limited the relevance of the data to the review's hypotheses. On the positive side, reviewing data from studies with different hypotheses can help to reduce the effect of publication bias on the overall picture. The findings that have been brought together indicate the potential utility of systematically investigating causal mechanisms that might connect depression with the persistence of positive psychotic symptoms.

It was not deemed appropriate to attempt a quantitative synthesis of the findings of the present review, due to the great heterogeneity of the included studies. A

quantitative understanding of the relationships of interest could be aided by the inspection of effect sizes obtained by relevant studies, which were not reported here.

As stated at the outset of this chapter, the practical aim of research such as that reviewed here is to guide the optimisation of therapies for people with psychosis. To this end, it will be necessary to understand further the specific causal processes that perpetuate symptoms. It is instructive that depression in people with psychosis often predicts the persistence of positive symptoms, but this may not be specific enough: what are the mechanisms of this association?

A multifactorial account of depression (taking in genetic, biochemical, cognitive, affective and social aspects) was accepted much earlier and acknowledged more widely (e.g. Baillie et al., 2009) than a similar approach to psychosis. In line with this, it is commonly held that depression can have many different causes, and furthermore that the treatment of it can be approached in several quite different ways, each of which has been shown to work (NICE, 2009), depending on the supposed mechanism of pathological persistence. Each treatment approach has its own merits, but none has been found to reliably benefit every patient. A comprehensive assessment of the predisposing, precipitating and perpetuating factors in each case can guide the application of appropriate techniques. This multifactorial complexity is likely also to apply to depression when it accompanies psychosis, and some of the maintenance factors might interact with psychosis more than others.

Some of the factors that prolong depression can be ameliorated with psychological therapy, and the main implication of this review is that these same mechanisms might have an effect on positive symptom persistence. Self esteem has received some attention as such a factor, and trials have reported that treatments targeting self esteem can have beneficial effects on positive symptoms. Appraisals of shame, loss and entrapment have not yet proved to be a beneficial therapeutic target for ameliorating depression (Jackson et al., 2009).

The main common pathway for the therapeutic effect of CBT (and some other therapies) on depression is thought to be cognitive change, that is reduction in negative thoughts and dysfunctional beliefs or assumptions (Coleman et al., 2009). Empirically supported mechanisms proposed to maintain negative cognition in depression include a tendency to ruminate in response to distress (Nolen-Hoeksema, 1991), systematic memory bias (Williams et al., 2007) and difficulties in solving interpersonal problems (Marx, Williams, & Claridge, 1992). As mentioned earlier in

this chapter, most of the cognitive processes associated with depression have not been systematically investigated in people with psychosis. It is very plausible that the same constellation of memory impairment, ruminative thinking style and problem solving difficulty that accompany depression (Williams et al., 2007) could make it very difficult for someone with delusions to engage with reality testing or even to effectively gather evidence to assess the validity of distressing beliefs. Given the promising results of trials targeting these features to treat depression, it would be of worth to find out whether the same processes can contribute to the persistence of positive symptoms, in which case treatment trials in psychosis would be indicated.

A prognostic study of the power of specific depression-related processes in predicting the persistence over time of individual psychotic symptoms could clarify the nature of these causal pathways. Given that delusions and paranoia show most consistent associations with depression, and that delusions of persecution are the delusion type associated with most negative affect (Appelbaum et al., 1999), persecutory delusions would appear to be a good candidate symptom for such an investigation. If effects are found here, it is possible that they might generalise to other psychotic symptoms.

2.9 Conclusions

A review of the available evidence found support for the hypothesis that high levels of depression in people with schizophrenia spectrum disorders are predictive of positive symptom persistence over time. Some studies using summed positive symptom scale totals did not find this predictive effect, but specific associations with delusions and paranoia were repeatedly seen.

The review's second hypothesis - that the effective treatment of depression in people with positive psychotic symptoms would be followed by reductions in positive symptoms as well as in depression - was partially supported. The effect depended on the modality of treatment. The addition of antidepressant medications to antipsychotic regimens did not appear to improve positive symptoms, at least not over periods up to 12 weeks. However, psychological therapies such as CBT that reduced depression in people with psychosis also often improved positive symptoms, and specific effects on delusions were found by every study that looked for them. The mechanisms of these effects, however, are unclear. Further investigation of these specific processes is indicated.

Garety, Freeman and colleagues' (2001) model was supported: the reports reviewed here are consistent with a causal role for depression-related processes in

perpetuating positive psychotic symptoms, particularly delusions. This does not contradict Birchwood's assertion that psychosis can be a cause of depression. The two types of problem are likely to mutually feed each other. The relatively consistent finding that self esteem interventions improved positive symptoms could be interpreted as supporting both Bentall et al.'s (2001) and Freeman et al.'s (2002) theories; the designs of the studies reviewed here did not provide a direct test of the contention regarding whether paranoia is a defensive or direct expression of underlying negative self-appraisals. Freeman et al.'s model may be considered the more parsimonious of the two.

If specific depression-related factors – processes that can be targeted in therapy – are found to predict the persistence over time of positive symptoms such as persecutory delusions, then targeted interventions could be trialled to help people who experience psychosis to recover more quickly. The next two chapters will identify prominent candidate processes in the depression literature, and make a case for their relevance to the maintenance of belief pathology.

Chapter 3. Depression and cognitive processes that might contribute to persistence of symptoms

In the preceding chapter, evidence was reviewed for a link between depression and the persistence of positive psychotic symptoms such as persecutory delusions. It was seen that an association has been found between depression and the persistence of such symptoms, and in particular of delusions and paranoia. In order to understand the meaning of this association and its implications for theory and clinical practice, it is important to examine the causal mechanisms of the relationship. This investigation can be informed by detailed psychological models of the persistence of depression, which have identified factors with probable causal significance. The same processes that perpetuate symptoms of depression might also contribute to the maintenance of psychotic belief pathologies.

The present chapter introduces the cognitive literature on symptom persistence in depression. Early theories emphasised the negative content of thoughts as a cognitive vulnerability for persistent depression (Teasdale, 1988; Beck, 1976). Some accounts focused specifically on the form of depressive thought processes, for example, repetitiveness or specificity, and the roles of these cognitive processes as responses to distress (Nolen-Hoeksema, 1991; Williams et al., 2007). An overview of these theoretical approaches is given here, and key cognitive constructs are picked out whose relevance has attracted empirical support.

3.1 Negative cognitive content: Beck and schematic beliefs

Judgements that people make about themselves and others synthesize past experience to influence subsequent perceptions and responses. Such beliefs may mediate the individual's adaptation to the social world (Fowler et al., 2006). Negative schematic beliefs are a central component of cognitive theories of depression (Beck, 1976; Clark, Beck, & Alford, 1999). Beck's original theory, in its simplest form, proposed that cognitive vulnerability to depression manifests as maladaptive schematic beliefs that drive a tendency to interpret certain life events in negative ways, causing low mood. Consistent with Beck's theory, depressed patients endorse more negative evaluations of the self, and have more dysfunctional attitudes, more depressogenic attributional biases and report more negative thoughts than do non-clinical controls (Bradley & Mathews, 1983; Ruchman, West, & Pasahow, 1985).

Beck's early account proposed that cognitive vulnerability to depression is a stable, enduring anomaly of one's psychological system (Beck et al., 1979, p. 20). The depressed person was conceptualised as qualitatively different from most people in their faulty or "primitive" inferential thinking style, having undergone a "cognitive revolution... which produces a marked *reversal* in the way the patient construes reality." (Beck et al., 1979, p21).

"According to this schematic representation, we observe that the depressed patient tends to view his experiences as total deprivations or defeats (nondimensional) and as irreversible (fixed). Concomitantly, he categorizes himself as a "loser" (categorical, judgmental) and doomed (irreversible character deficits)." (Beck et al., 1979, p15).

This proposition of a relatively stable cognitive vulnerability, which should be observable in depression-prone individuals at any point in time, has not been consistently supported by empirical research: studies have typically found elevated depressive cognitive markers in people only during an episode of depression and not when they have recovered from such an episode (even when no psychological therapy was involved in their recovery), when the underlying cognitive vulnerability should presumably still be present (Hamilton & Abramson, 1983; Silverman, Silverman, & Eardley, 1984; Simons, Garfield, & Murphy, 1984). Negative thinking is clearly not without causal relevance in the perpetuation of depression, but may not be a stable feature of depression-prone individuals.

Another result of the relatively stable conceptualisation of vulnerability put forward by Beck in his early account is that the theory does not explain why most people who experience a depressed state soon return to normal mood even without treatment, while others get increasingly depressed and become trapped in a chronic cycle of disorder (Depue & Monroe, 1986). Limitations like these led investigators allied with the cognitive psychological approach to develop more elaborate, multifactorial and reactive perspectives on the mental processes involved in depression, which Beck would in turn incorporate into his later expanded model (Beck, 2008).

3.2 The distinguishing features of persistent depression

Targeting negative automatic thoughts and dysfunctional assumptions in cognitive therapy can be very effective, leading to improvements in mood and reductions in negative thinking (Teasdale & Fennell, 1982; Gloaguen, Cottraux, Cucherat, & Ivy-Marie, 1998; Keith S., 1989). However, there is a substantial group of people who do not benefit from this therapeutic approach. The relatively more treatment resistant

conditions of such people might be complicated by some particular features that make the depression more severe or tenacious. Moore & Garland (2003) propose that persistent chronic depression can be distinguished from that which follows a short episodic course in several ways, each of which has relevance for theory and treatment.

Firstly, persistent depression is characterised by avoidance: behavioural withdrawal, as well as mental avoidance of upsetting thoughts and of painful emotions. Avoidance strategies start off by enabling the individual to cope with extremely aversive circumstances and to limit acute distress on a day-to-day basis, but can be ultimately maladaptive when overused with the intention of eliminating unpleasant experience. Habitual avoidance can contribute to an intolerance of upsetting emotions, a lack of reinforcement from the environment and poor problem solving skills that have not developed due to non-engagement with difficult issues. Secondly, persistent depression is often characterised by an extreme categorical thinking style, where attitudes such as “I fail at every thing that I attempt” become abstracted from any specific memories and generalised beyond reason. This thinking style can interfere with flexible coping and with constructive problem solving. Third, Moore & Garland propose that the underlying beliefs of people with persistent depression are particularly extreme. Negative core beliefs, conditional assumptions and beliefs about depression can all contribute to the perpetuation of the low mood state. Finally, social factors at all stages of the disorder’s development can affect its trajectory: early experiences can predispose to more extreme pathology, stressful or traumatic events can trigger its manifestation, and ongoing features of the social setting of the depressed person, such as an unrewarding job or unsupportive relationships might maintain emotional difficulties.

Cognitive accounts of the persistence of depression typically show a significant degree of overlap in the set of processes that they deem to be important in symptom maintenance. One of the earliest cognitive theories to tackle persistence specifically from an information processing point of view was Teasdale’s differential activation hypothesis.

3.3 Teasdale’s differential activation hypothesis

John Teasdale extended an account, based on Beck’s cognitive theory of depression (Beck et al., 1979), which dealt specifically with the maintenance of symptoms, rather than their aetiology (Teasdale, 1988). Teasdale proposed that the distinguishing feature of persistent depression, as opposed to mild or transient depressive states, is

that negative thoughts and low mood become coupled in a mutually exacerbating downward spiral. This same cognitive maintenance cycle had been noted in Beck's original account. The differential activation hypothesis sought to explain why some people and not others can become locked in such a vicious cycle.

Teasdale proposed that cognitive vulnerability to the persistence of depression depends on the nature of thoughts that *become accessible in a low mood state*: these will determine whether or not a vicious cycle of lower mood and more negative cognition becomes established. The theory differs from Beck's in that the vulnerability is reactive – it lies in the nature of the person's response to a low mood state - and thus would not necessarily be observable when the person is in good spirits. Consistent with this proposal, those with a tendency towards depression respond to experimentally induced low mood with more self-devaluative thoughts and feelings than those who have never been depressed (Teasdale & Cox, 2001), and among people who are depressed, those with more negative thoughts and beliefs recover more slowly than those who think less negatively (Dent & Teasdale, 1988; Williams, Healy, Teasdale, White, & Paykel, 1990).

3.4 The role of memory

Both Beck's and Teasdale's theories centrally concern the negative content of cognition, in the form of memories, beliefs and attitudes. However, the theories differ in the emphasis they place on these different cognitive constructs. Beck saw maladaptive schematic beliefs about the self and others, probably formed during childhood, as the fundamental drivers of unhelpful attitudes and negative thoughts. Teasdale wrote more about memory. The differential activation hypothesis was based on associative network theories of memory and affect, such as that of Gordon Bower (1981), which are rooted in the experimental cognitive psychology tradition. Associative network theories see cognitive systems as networks of nodes, where each node has a meaning and connections of various strengths with nodes corresponding to concepts that it has previously been paired with. Thus when a node becomes activated to a certain degree, activation can spread to connected nodes proportionately to the strength of the connections. The context-dependent learning phenomenon, whereby material memorised in a particular environment is recalled more readily in the same environment than a different one (e.g. Godden & Baddeley, 1975), lends itself well to an associative network explanation. The reactivation of a context node here leads to spreading activation, raising the likelihood that nodes corresponding to items that have been encountered in this context will become active. Bower's "network theory of affect" proposed that an emotional state could serve as

context in such a model, and thus drive state-dependent encoding and retrieval of memories (Blaney, 1986). In support of this proposal, information encoded in a state of low mood is subsequently more accessible when mood is again low than are memories from happier states (Bower, Monteiro, & Gilligan, 1978; Singer & Salovey, 1988). Mood congruent memory is a related phenomenon: negative information is learned more readily in low mood than positive information, and recalled more quickly. Matt et al.'s (1992) review of mood congruency studies in relation to depression concluded that while non-depressed people recalled 6%-8% more positive than negative stimuli, clinically depressed participants showed the opposite bias: up to 10% more negative memories than positive ones. Given that the activation of mental representations of unpleasant events or bad experiences can affect mood in the same way as exposure to the events themselves, it is consistent that interactions of mood with information processing in memory could contribute to the vicious cycle that Teasdale describes in persisting depression. According to the differential activation hypothesis, someone with a large amount of negative content stored in memory will, when in low mood, experience the associative activation of this volume of unpleasant material, and will thus be more susceptible to a mutually reinforcing cognitive-affective downward spiral than someone with more positive memories.

3.4.1 Overgeneral autobiographical memory

As well as the valence of the content, the *form* of autobiographical memory appears to be different in people with depression to those without. Williams & Broadbent (1986) were the first to discover an overgenerality bias in the memories of people with depression. They studied 25 patients in the first few days after hospitalisation for a suicide attempt (most of whom had been experiencing severe depression). Participants were asked to recall a specific autobiographical memory in response to each of a set of positive and negative cue words (e.g. "happy", "angry"). Control participants tended to produce a specific memory, as instructed (e.g. "last Tuesday evening, I was happy when I went to the cinema with my sister"), but there was a striking tendency for overdose patients to report instead generalised memories (e.g. "seeing my sister makes me happy"). This effect, observed for both positive and negative cue words, was named overgeneral autobiographical memory bias (OGM).

These findings have since been replicated and extended using variations of Williams and Broadbent's original task, which has come to be known as the Autobiographical Memory Task (AMT). A number of studies by different research groups have shown OGM in adults with clinical depression (Brewin, Watson, McCarthy, Hyman, & Dayson, 1998; Goddard, Dritschel, & Burton, 1996; Kuyken & Dalgleish, 1995; Puffet,

Jehin-Marchot, Timsit-Berthier, & Timsit, 1991; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001), adolescents with major depressive disorder (Park, Goodyer, & Teasdale, 2002) and women with postnatal depression (Croll & Bryant, 2000), as well as in currently euthymic individuals with past major depressive or bipolar episodes (Mackinger, Pachinger, Leibetseder, & Fartacek, 2000; Scott, Stanton, Garland, & Ferrier, 2000) and in sub-clinically dysphoric participants (Goddard, Dritschel, & Burton, 1997; Ramponi, Barnard, & Nimmo-smith, 2004). This combination of findings suggests that OGM could be both a trait and a state marker of depression.

Empirical research findings support and somewhat extend Moore & Garland's (2003) observation that an overgeneral categorical thinking style is most pronounced in people with particularly persistent depression. Brittlebank and colleagues found that OGM in a sample of 22 depressed patients predicted the persistence of depression at 7-month follow-up (Brittlebank, Scott, Williams, & Ferrier, 1993). Similar findings were obtained by Peeters et al. (2002) and by Raes et al. (2006). This suggests the possibility of a causal association. A specifically targeted intervention, Memory Specificity Training (MEST), has shown promising effects in a preliminary investigation with depressed inpatients (Raes et al., 2009).

3.5 Responses to distress

Both Teasdale's and Williams' theories have drawn on aspects of Susan Nolen-Hoeksema's work on rumination. The Response Styles Theory (Nolen-Hoeksema, 1991) concerns a person's *responses* to low mood, and how these can affect its subsequent course. Some people might respond to low mood by suppressing the thought of it, some by distracting themselves with activities, some might seek social support, and some might ruminate on the aversive state.

3.5.1 Rumination

Rumination has been defined as "repetitive and passive thinking about one's symptoms of depression and the possible causes and consequences of these symptoms" (Nolen-Hoeksema, 1991). Rumination is related to worry, in that both constructs involve a repetitive negative thought process, but they vary in content: worry anticipates future threat, while rumination is more closely related to past loss and failure (Segerstrom, Tsao, Alden, & Craske, 2000; Watkins, Moulds, & Mackintosh, 2005). Rumination is usually more closely associated with depression, and worry with anxiety (McLaughlin, Borkovec, & Sibrava, 2007).

The tendency to ruminate in response to distress appears to be a relatively stable individual difference characteristic (Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Just & Alloy, 1997). It is typically measured by questionnaire, for example the Ruminative Response Scale of the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991; Just & Alloy, 1997) or the Rumination on Sadness Scale (Conway, Csank, Holm, & Blake, 2000). The Response Styles Theory proposes that rumination prolongs episodes of depression by amplifying the effects of low mood on negative thinking and allowing depression to interfere with problem solving and instrumental behaviour (Nolen-Hoeksema, 1991). Prospective studies show that a ruminative response style predicts the onset of depression (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker, & Larson, 1994; Spasojević & Alloy, 2001). Rumination can also predict both the severity and duration of depression (Nolen-Hoeksema, 2000). For example, Raes et al. (2006) found that rumination in a sample of 28 depressed patients was a significant predictor of depression severity 7 months later, even after controlling for baseline depression severity.

A ruminative thinking style can be induced experimentally in the short term, which negatively affects mood (Donaldson & Lam, 2004; Nolen-Hoeksema & Morrow, 1993). Rumination-focused cognitive behavioural therapy, developed to target this process in the long term, has demonstrated encouraging preliminary results in a case series of 14 individuals with treatment-refractory residual depression (Watkins et al., 2007). Rumination reduced significantly following up to 12 sessions of therapy, and 50% of the patients treated achieved full remission (defined as a score below 8 on the HRSD at termination and below 9 on the BDI for 4 consecutive weeks).

3.5.2 Avoidance

Avoidance of several kinds has been implicated in the persistence of depression. Moore & Garland (2003) distinguished three types.

1. Behavioural avoidance. Avoidance of activities can be manifest as gross withdrawal, or in more subtle forms such as avoidance of deviation from a routine.
2. Cognitive avoidance. This relates to unwanted thoughts, memories or discussions of particular situations.
3. Emotional avoidance, of painful affect.

Behavioural avoidance was the first of these to receive much attention in the literature (Mowrer, 1939) as a mechanism by which access to environmental reinforcers, which might usually have alleviated a transient state of depression, becomes restricted, perpetuating a pathologically low mood state. Behavioural Activation therapy

(Lewinsohn, Antonuccio, Steinmetz, & Teri, 1984) was developed to correct this loss of positive reinforcement, and to this day shows positive effects on people with depression which some argue are equivalent to the efficacy of more complex cognitive therapy packages (Jacobson & Gortner, 2000). Leventhal (2008) argued the case that avoidance behaviour is the central cause of the transformation from “normal” states of sadness to pathological depression.

The internal forms of avoidance described by Moore & Garland can be subsumed under the construct of “experiential avoidance” proposed by Steve Hayes (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Experiential avoidance is defined as “the phenomenon that occurs when a person is unwilling to remain in contact with particular private experiences (e.g. bodily sensations, emotions, thoughts, memories, behavioural predispositions) and takes steps to alter the form or frequency of these events and the contexts that occasion them, even when these forms of avoidance cause behavioural harm” (Hayes et al., 2004; Hayes et al., 1996). Bringing together the findings from studies totalling 1068 mental health service users and 1347 university students, Hayes et al. (2004) report that higher levels of experiential avoidance were associated with higher levels of both depression and anxiety, as well as trauma, general psychopathology and a lower quality of life. These associations have been widely replicated in general population samples and groups with affective disorders (see Chawla & Ostafin (2007) for a review). Acceptance and commitment therapy (Hayes, Strosahl, & Wilson, 1999) is one of a number of “third wave” psychological treatments that explicitly targets avoidance (Hayes, Jacobson, Follette, & Dougher, 1994), and this approach has shown significant positive impact on depression in trials (Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Zettle & Rains, 1989).

3.6 Theoretical integration

The cognitive processes invoked in the accounts above are consistently shown to be interrelated, and can comprise a multifactorial dynamic account of the persistence of symptoms in depression. Mark Williams proposed the Capture and Rumination, Functional Avoidance and eXecutive control model (CaR-FA-X; Williams, 2006) to integrate the overgeneral memory bias phenomenon within a framework of related processes, including difficult experiences, functional avoidance, ruminative thinking and problem solving difficulties. A diagram of the model is reproduced in Figure 3.1.

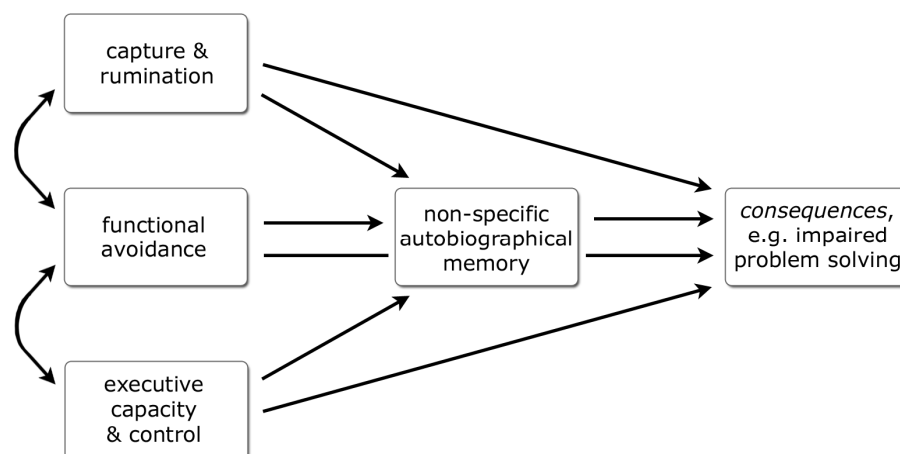


Figure 3.1. The CaR-FA-X model, reproduced from Williams (2006).

The construct of functional avoidance invoked here is very similar to Hayes' description of experiential avoidance: functional avoidance is the process whereby individuals avoid re-experiencing specific painful memories of negative past events because of the distress that they cause. Williams describes a process of memory retrieval in which the search for a specific episode gets truncated at an intermediate (overgeneral) stage. This truncation is reinforced as it serves to avoid the negative affect associated with specific bad memories. In this way "functional avoidance" connects negative cognitive content (as emphasised by Beck and Teasdale) with the development of an enduring overgenerality in autobiographical memory. Ruminative processing can "capture" the retrieval process at this intermediate abstract-conceptual level, further decreasing the likelihood of a specific memory being retrieved. The capacity for executive control affects the individual's ability to remain focused on retrieval in the face of distraction.

Consistent with the CaRFAX model, it has been shown that autobiographical memory task performance correlates with scores on measures of avoidant coping (Hermans, Defranc, Raes, Williams, & Eelen, 2005), rumination (Raes et al., 2005) and executive control (Dalgleish et al., 2007). Further, experimental studies have shown that induction of a ruminative thinking style reduces memory specificity, both in depressed patients (Watkins & Teasdale, 2004; Watkins & Teasdale, 2001) and in students (Raes, Watkins, Williams, & Hermans, 2008). Rumination also impairs effective problem solving (Donaldson & Lam, 2004). Interference with problem solving ability is a common pathway by which all of the theories outlined above propose that depressive cognitive biases exert their effects on symptom persistence (Beck, 1976;

Teasdale, 1988; Nolen-Hoeksema, 1991; Williams, 2007). Problem solving difficulties are thought to perpetuate dysfunction by impairing instrumental behaviours and leading the individual into unhelpful situations.

3.6.1 Problem solving difficulties

Depressed people often have difficulty in solving interpersonal problems (Beck, 1976). Marx et al. (1992) established that patients with depression perform more poorly than healthy controls on a measure of social problem solving cognition, the Means-Ends Problem Solving task (MEPS; Platt & Spivack, 1975). In this task, participants are presented with the beginning and the end of each of a set of stories. Each beginning describes the protagonist with a particular need, and each ending shows the need having been met. The task requires the participant to provide the middle of the story, in the form of a sequence of means. Responses are scored on the number of relevant means produced and the overall effectiveness of the solution.

Poor problem solving performance is not specific to depression: in Marx et al.'s (1992) study, a group of anxious patients also performed more poorly than controls. The problem solving deficit measured by the MEPS may be a general psychopathological marker. Platt & Spivack's early studies described MEPS deficits in a mixed group of psychiatric inpatients dominated by people with psychotic disorders (Platt & Spivack, 1972a; Platt & Spivack, 1972b).

Problem solving performance prospectively predicts recovery from depression. For example, Garland et al. (2000) found that problem solving performance predicted 3-month and 6-month prognosis in a group of 20 depressed patients. Problem solving therapy focuses on training in adaptive problem solving attitudes and skills. A recent meta-analysis of studies with 21 independent samples concluded that problem solving therapy is significantly more effective in reducing depressive symptoms than no treatment or support groups, and equally as effective as other psychological therapies or medication (Bell & D'Zurilla, 2009).

3.6.2 Summarising empirically supported cognitive factors in depression persistence

Several features stand out in the literature as reliably associated with symptom persistence in depression, and also as promising therapeutic targets where intervention might interrupt the vicious cycle of dysfunction. These are: depressogenic cognitive content, in the form of negative memories, dysfunctional beliefs and attitudes; an overly general bias in autobiographical memory; the tendency to

ruminate in response to distress; avoidance; and difficulties with interpersonal problem solving. In conceptualising ways in which coexisting depression might lead to the persistence of psychotic symptoms such as delusions, these cognitive features are potentially relevant. In the next chapter, existing evidence will be reviewed for the associations of these depression related cognitive factors with psychosis, and the case will be examined for a systematic investigation of the role that these processes might play in the maintenance of persecutory delusions.

Chapter 4. Depression related cognitive processes and persecutory delusion persistence

The previous chapter identified cognitive constructs that stand out in the literature as linked with the persistence of depression. These processes have not yet been investigated together in a group of people with persecutory delusions, although several have been examined individually, with some evidence suggesting causal effects. The research concerning depression-related cognitive factors in people with persecutory delusions is summarised in this chapter, which concludes with a proposed framework for their interactive roles in the maintenance of distressing threat beliefs.

4.1 Schematic beliefs

Negative core beliefs about the self and others have been emphasised in recent cognitive models of persecutory delusions (Bentall et al., 2001; Bentall & Swarbrick, 2003; Freeman et al., 2002). Persecutory delusions have been argued to relate to such beliefs (e.g. “I am vulnerable”; “other people are devious”) in a direct and non-defensive way (Smith et al., 2006; Freeman et al., 2002). Research by Freeman, Fowler and colleagues has been consistent with this proposal. Fowler et al. (2006) found that a sample of 252 people with psychosis endorsed significantly more negative schematic beliefs both about self and about others than a non-clinical control group. In a related study, employing an overlapping sample, Smith et al. (2006) found, among a sample of 100 psychosis patients, that those endorsing more negative beliefs about self and others had persecutory delusions of greater severity and were more preoccupied and more distressed by them than those endorsing less negative beliefs.

Fowler et al. (2011) extended these cross-sectional findings, using structural equation modelling to examine the longitudinal relationships between negative cognition, depression and paranoia over a year in 301 people with psychosis. Models where the direction of effect went from negative cognition and depression to paranoia were found to fit the data significantly better than models in which paranoia led to negative cognition and depression, suggesting that paranoia is causally affected by these processes. The “negative cognition” factor used here was a combination of the “negative beliefs about self” subscale of the Brief Core Schema Scales (BCSS; Fowler et al., 2006) and the negative cognition subscale of the Beck Depression Inventory

(BDI-II; Beck, Steer, & Brown, 1996), but the authors reported that using only BCSS items for the negative cognition factor produced the same pattern of results.

A contrasting view is that persecutory ideation is the result of defence mechanisms acting to preserve fragile self esteem (Bentall et al., 1994). People with persecutory delusions show an externalising attributional bias for negative events, particularly towards blaming other *people* for bad things happening (reviewed in Bentall et al., 2001; Garety & Freeman, 1999). However, Bentall and colleagues' proposal that this attributional bias functions to protect self esteem, camouflaging implicit negative beliefs about the self with explicit normal ones by deflecting the blame for negative events onto others (Bentall et al., 1994; Bentall & Kaney, 1996), has been controversial. A defence account would be consistent with relatively high or normal levels of self esteem in patients, whereas low self esteem has frequently been reported (e.g. Bentall et al., 2008; Fowler et al., 2011; Freeman et al., 1998). Garety & Freeman's comprehensive review (1999) also failed to find consistent support for implicit-explicit self esteem discrepancies in paranoid individuals. These mixed findings may have to do with difficulty in reliably measuring self esteem, particularly implicit self esteem, or it may be that self esteem is particularly variable over time in people with persecutory delusions (Bentall et al., 2001). Whether or not defensive processes are invoked, these theories agree on the importance of negative appraisals of the self in driving paranoid thinking.

As discussed in more detail in Chapter 2, several trials have used self esteem interventions for people with psychosis (Hall & Tarrier, 2003; Knight et al., 2006; Laithwaite et al., 2009; Laithwaite et al., 2007), and most of these achieved concurrent reductions in depression and in positive psychotic symptoms. This convergent evidence supports the proposal that negative beliefs about the self can contribute to the persistence both of depression and of delusional persecutory beliefs.

4.1.1 Developmental origins of schematic beliefs

Schematic belief structures are thought to mediate the effect of early adverse life experiences on the later development of psychopathology. It is well established that early loss and trauma events predict the development of later emotional and psychological problems, including affective disorders and psychosis (e.g. Cirulli, Berry, & Alleva, 2001; Read, Perry, Moskowitz, & Connolly, 2001; Heim, Owens, Plotsky, & Nemeroff, 1997). However, most individuals who have adverse experiences in childhood do not go on to develop significant psychopathology: adversity is not a sufficient condition. In those who do, it is thought that the experience

of poor attachment relationships is internalised and carried forward into adulthood in the form of internal representational models which go on to influence maladaptive behaviour, increasing vulnerability to adult psychopathology (Bowlby, 1988).

The formulation of mental models of the self and others given in the developmental attachment literature can be seen as equivalent to the construct of schematic beliefs such as are measured by Fowler's Brief Core Schema Scales, which have shown a strong association with persecutory ideation (Fowler et al., 2006; Fowler et al., 2011). The conceptualisation of internal working models put forward in the attachment literature is distinguished from Beck's original description of the schema concept in that it reflects "more motivated and affectively charged constructs, representing emotional states associated with interpersonal relationships as well as beliefs" (Berry, Barrowclough, & Wearden, 2007, p. 465). The broad developmental perspective of attachment theory can be helpful in conceptualising the interactive contributions of cognitive, affective and interpersonal factors to the persistence of symptoms such as paranoia.

4.1.2 Attachment theory and psychosis

A long-established body of research on infants has demonstrated that the majority very quickly (within the first year of life) develop one of three broad patterns of attachment behaviour towards a primary caregiver: secure attachment, insecure ambivalent attachment, or insecure avoidant attachment (Ainsworth, Blehar, Waters, & Wall, 1978). Secure infants use the primary caregiver as a base for exploration, show signs of missing the caregiver when parted, and are comforted readily when the caregiver returns. Insecure ambivalent infants are often too fretful to explore, become very unsettled when parted from the primary caregiver, and show anger or excessive distress when reunited. Insecure avoidant infants explore readily, show little sign of distress when parted from the caregiver, and actively avoid contact upon the caregiver's return. Attachment styles tend to be relatively stable throughout an individual's life, and appear to transmit through generations (Waters, Merrick, Treboux, Crowell, & Albersheim, 2000; Benoit & Parker, 1994). Securely attached children are likely to become autonomous adults, while ambivalent children develop preoccupied or enmeshed adult attachment behaviours, and avoidant infants grow up to be dismissive of attachment.

Groups of people with psychosis (including chronic and first episode samples) show a high rate of insecure attachment styles (Dozier, Cue, & Barnett, 1994; Couture, Lecomte, & Leclerc, 2007; Dozier & Lee, 1995; Dozier, Stevenson, Lee, & Velligan,

1991), particularly of the avoidant type (MacBeth, Gumley, Schwannauer, & Fisher, 2011; Ponizovsky, Nechamkin, & Rosca, 2007; Berry, Barrowclough, & Wearden, 2008) (reviewed in Berry et al., 2007; Read & Gumley, 2008). Data are scarce regarding specific relationships of attachment styles with individual symptoms, but there is some evidence of a significant association between insecure attachment and paranoia (Berry, Wearden, Barrowclough, & Liversidge, 2006; Berry et al., 2008; MacBeth, Schwannauer, & Gumley, 2008). It has mostly been reported that insecure attachments are associated with a broad range of symptoms, but a recent analogue study (Pickering, Simpson, & Bentall, 2008) found among 503 students that an insecure attachment style specifically predicted paranoia, not hallucinations. It has been proposed that insecure attachment can lead to paranoia and interpersonal distrust via underdeveloped capacities to self-soothe, to attune to the mental states of others and to reflect on one's own affective experience, and through the use of submissive/subordination or dominant interpersonal strategies (Gumley & Schwannauer, 2006).

4.2 Avoidance

Moore & Garland (2003) distinguished behavioural, cognitive and affective forms of avoidance in relation to persistent depression. Each of these has been examined, albeit to a lesser extent, in people with psychosis.

4.2.1 Behavioural avoidance

Behavioural avoidance, as a safety-seeking behaviour, is known to be very common in people with persecutory delusions. Freeman et al. (2001) found that 23 out of 25 participants with persecutory delusions had used the strategy of avoidance in the last month, and Freeman et al. (2007) similarly found this for 78 out of 100 participants. It is hypothesised that avoidance and other related safety behaviours, such as escape from situations of perceived threat, contribute to the persistence of delusional beliefs by preventing contact with disconfirmatory evidence: if you avoid or always escape from a feared situation, you are unlikely to find out that it might actually be harmless (Freeman, 2007). This formulation was influenced by cognitive accounts of the persistence of anxiety disorders (e.g. Salkovskis, 1991), and behavioural avoidance has since become an established therapeutic target in the psychological treatment of persecutory delusions.

4.2.2 Experiential avoidance

Cognitive and emotional avoidance, subsumed under the construct of experiential avoidance, has been conceptualised as a central process in the development and maintenance of psychological distress across the diagnostic spectrum (Hayes et al., 2004), and recently this has received particular attention in relation to delusions and paranoia. Goldstone et al. (2011) found that experiential avoidance (EA) statistically fully mediated the cross-sectional relationship between life hassles and both delusions and delusional distress in a group of 100 people with psychosis, and that it also partly mediated this relationship in a non-clinical control group. Two more non-clinical studies have reported similar findings. Udachina et al. (2009) found with an experience sampling method that current high EA in subclinically paranoid students predicted subsequent increases in paranoia, even when previous paranoia was taken into account. Oliver et al. (2011) reported that among 700 university students, psychological flexibility – the direct inverse of EA, measured in the same way – moderated the association between negative schematic beliefs and delusional thinking (the association was also mediated by anxiety).

4.2.3 Therapies targeting avoidance

Acceptance and Commitment Therapy (ACT; Hayes et al., 1999), which targets EA specifically as a means of changing the way that clients relate to distressing experiences, has been applied to psychosis in three randomised controlled trials (Gaudiano & Herbert, 2006; Bach & Hayes, 2002; White et al., 2011). Findings included lower psychotic symptom believability after ACT compared to treatment as usual (Bach & Hayes, 2002; Gaudiano & Herbert, 2006), fewer hospital admissions (Bach & Hayes, 2002) or crisis contacts (White et al., 2011), more reductions in negative affect or depression (Gaudiano & Herbert, 2006; White et al., 2011), in the distress associated with psychotic symptoms (Gaudiano & Herbert, 2006) and in negative symptoms (White et al., 2011). Mindfulness based cognitive therapy (MBCT; Segal, Williams, & Teasdale, 2002) also seeks to reduce clients' avoidant struggle with symptoms, the focus here being non-judgmental awareness of experience. MBCT has established efficacy for relapse prevention and symptom reduction in recurrent depression (Teasdale et al., 2000; Helen Ma & Teasdale, 2004; Hofmann, Sawyer, Witt, & Oh, 2010; Barnhofer et al., 2009), and recent evidence suggests that it can also benefit people with psychosis (Chadwick, Taylor, & Abba, 2005; Chadwick, Hughes, Russell, Russell, & Dagnan, 2009). These treatment studies add weight to the hypothesis that avoidance, as well as having a special relationship with depression, is also likely to be an important process in the pathological persistence of positive psychotic symptoms like delusions.

4.3 Overgeneral Autobiographical Memory (OGM)

The overgeneral autobiographical memory effect has not been systematically investigated in relation to delusions, and the findings of the few relevant studies are inconsistent. Williams et al.'s (2007) comprehensive review of research concerning autobiographical memory specificity and emotional disorder found the strongest links of OGM with depression and with trauma, but also included a study of people with persecutory delusions (Kaney, Bowen-Jones, & Bentall, 1999) and another of post-psychotic depression (Iqbal, Birchwood, Hemsley, Jackson, & Morris, 2004). Kaney et al. (1999) compared the autobiographical memory task (AMT; Williams & Broadbent, 1986) performance of 20 inpatients with delusions, 20 depressed outpatients and 20 non-clinical controls, hypothesising that people with delusions would show similar information processing biases to those who are depressed. The delusions group did show an overgeneral memory bias compared to controls, but surprisingly, the depressed group in this study did not show a statistically significant OGM effect. This finding is difficult to interpret. Given that this was the first study of OGM in people with persecutory delusions, the results might indicate that the memory bias is even stronger in this group than in people with non-psychotic major depression. However, the contribution of comorbid depression in the delusions group was not investigated in relation to OGM. These 20 patients had an average BDI of 14.7 (in the “mild depression” range) with a standard deviation of 9.57, and the authors report no analysis of the relationship between BDI scores and AMT performance. The OGM effect may or may not have been related to comorbid depression, or, as the authors suggest, to trauma history.

Iqbal et al.'s (2004) study concerned post-psychotic depression occurring in the wake of a first psychotic episode. They compared 13 first-episode patients who had developed this form of depression to a group of 16 who had not, and found that the depressed group produced fewer specific memories on the AMT. The nature of the participants' psychotic symptoms at the time of testing was not reported. Taylor et al (2010) recently reported the counterintuitive finding that among a group of 60 people with schizophrenia, a history of suicide attempts was associated with *good* performance on the AMT – *specific* memories. Notably, this effect was observed when adjusting for depression and trait anxiety: suicide attempts are associated with memory specificity *within* a particular level of affective disturbance. The authors propose that memory overgenerality may serve a protective function for people with psychosis in restricting access to the content of painful experiences. Any association between OGM and depression within the group was not reported. The study also, like Iqbal et al. (2004), did not report on the participants' psychotic symptoms, so the

relevance of the findings to persecutory delusions is questionable. A more thorough investigation of the potential role of OGM in delusion persistence should pay due attention to its strong association with potentially coexisting depression.

4.4 Rumination and worry

Rumination has hardly been examined in people with persecutory delusions, the only available report being of a pilot intervention study (Hepworth et al., 2011) targeting increased emotional processing. Significant reductions were found from pre- to post-treatment in delusion severity, depression and worry, but the reduction in rumination was non-significant. The scores of the participants with delusions on the ruminative response scale (RRS) of the response styles questionnaire (Nolen-Hoeksema & Morrow, 1991) were very high at both assessment points: mean scores were 58.58 pre-therapy and 49.83 post-therapy. For comparison, Donaldson & Lam (2004) found mean RRS scores of 34.2 in a group of non-psychotic depressed patients and 14.2 among non-clinical controls.

Worry – a construct closely related to rumination, sharing the form of negative repetitive thought – has received some attention in people with persecutory delusions. Groups with persecutory delusions can have very high levels of worry, comparable to those seen in generalized anxiety disorder (Freeman & Garety, 1999; Bassett, Sperlinger, & Freeman, 2009). Levels of worry are associated with intensity of delusional distress (Morrison & Wells, 2007; Freeman & Garety, 1999), and worry has been shown to predict the persistence of delusions over time (Startup et al., 2007). Furthermore, a randomised controlled trial of a psychological intervention reducing worry in people with persecutory delusions also achieved significant reductions in delusional distress and a trend towards reductions in the frequency of paranoid thoughts (Foster et al., 2010).

Hepworth et al. (2011) proposed that worry and rumination exert a negative impact on delusions through a detrimental impact on emotional processing. This pathway of effect through avoidance of emotional processing has been established in the context of depression and anxiety disorders (Sibrava & Borkovec, 2006; Watkins, 2004). Worry and rumination are dominated by verbal linguistic processing, which can prevent full activation of emotional responses (Vrana, Cuthbert, & Lang, 1986) and thus interfere with habituation to the distressing content (Foa & Kozak, 1986). Both types of repetitive thinking are of interest with respect to their associations with delusion severity and persistence.

4.5 Problem solving Difficulties

Poor problem solving performance is characteristic of people with depression, but is not specific to this disorder. The deficit measured by the Means-Ends Problem Solving task (MEPS; Platt & Spivack, 1975) may be a general psychopathological marker. In Marx et al.'s (1992) study, for example, both groups of depressed and anxious patients performed more poorly than controls. Notably, Platt & Spivack's early studies described MEPS deficits in a mixed group of psychiatric inpatients dominated by people with psychotic disorders (Platt & Spivack, 1972a; Platt & Spivack, 1972b). There is also some evidence that problem solving therapy (Bell & D'Zurilla, 2009) can lead to improvements in psychotic symptoms (Tarrier et al., 1993), though a specific relationship with persecutory delusions has not been investigated.

Mark Williams' CaRFAX model (Williams, 2006), as described in the previous chapter, conceptualises problem solving difficulties as a common mediator through which more specific cognitive factors such as memory bias can exert their effects on symptom persistence. It is likely that cognitive processes independent of affect can also impair problem solving performance. The CaRFAX model acknowledges here the contribution of overall executive capacity (Kyllonen & Christal, 1990; Dalgleish et al., 2007). No associations have been found between performance on the MEPS and IQ in groups of depressed and non-depressed students (Gotlib & Asarnow, 1979), but it remains possible that this relationship could exist in people with delusions.

Particular psychosis-related cognitive features may also be relevant to the problem solving performance of groups of people with persecutory delusions. The "jumping to conclusions" (JTC) reasoning bias (Garety et al., 1991) is a data gathering bias whereby individuals seek fewer pieces of evidence than control groups would before coming to accept a given belief. JTC is reliably found in people with persecutory delusions (e.g. Startup et al., 2008; Corcoran et al., 2008), and is relatively independent of affect (Fine, Gardner, Craigie, & Gold, 2007; So, Freeman, & Garety, 2008): this reasoning bias is thought to contribute to delusional severity independently of the influence of emotional dysregulation (Garety et al., 2005). The relationship between JTC and social problem solving performance has yet to be systematically examined.

4.6 Theoretical integration

Freeman et al.'s (2002) model, as described in Chapter 1, explicitly implicated depression related cognitive factors in the persistence of persecutory delusions, via

the gathering of biased evidence to support the threat belief, but details of the specific processes involved and the mechanisms of their effects have so far been unclear. The preceding chapter reviewed the systematic body of work conducted in the field of depression by Mark Williams' research group, and others, regarding cognitive mechanisms of symptom persistence, and in the present chapter, insights from depression research were applied to elaborating our understanding of the key transferable cognitive features that might also influence the persistence of persecutory delusions.

Williams' (2006) CaRFAX model of depression persistence, as introduced in Chapter 3, describes a number of specific biased cognitive processes and a framework conceptualising their maladaptive effects via difficulties in social problem solving. Figure 4.1 depicts a hypothetical hybrid model, in which the details of these cognitive processes and their effects, as described in the depression literature, are applied to flesh out the part of Freeman et al's (2002) model that concerns the effect of depressive cognitive factors on the gathering of evidence to support threat beliefs (which is hypothesised to contribute to their persistence over time).

The framework depicted in Figure 4.1 is specifically concerned with features that are known to be related to depression; factors that are relatively independent of affect, such as executive capacity and JTC, are not included here. Negative schematic beliefs are thought to develop through difficult early experiences; they are relatively stable, and by their unpleasant nature can provoke maladaptive coping responses such as rumination and/or avoidance. These response strategies prevent contact with specific details of lived experience, and thus can result in an overgeneral autobiographical memory bias. Effective social problem solving is impaired by the scarcity of specific information accessible in memory, as well as by avoidance of certain experiences and by ruminative preoccupation of thought. Poor problem solving is likely to increase the occurrence of negative experiences, which can reinforce the threat belief, ruminative thinking may cause the over-representation of negative thoughts through repetition, and an overgeneral style of memory could reinforce the rigidity of the belief by failing to retain the nuances of specific events. Thus it is plausible that the same processes that maintain depression could also contribute to the persistence of distressing persecutory delusions.

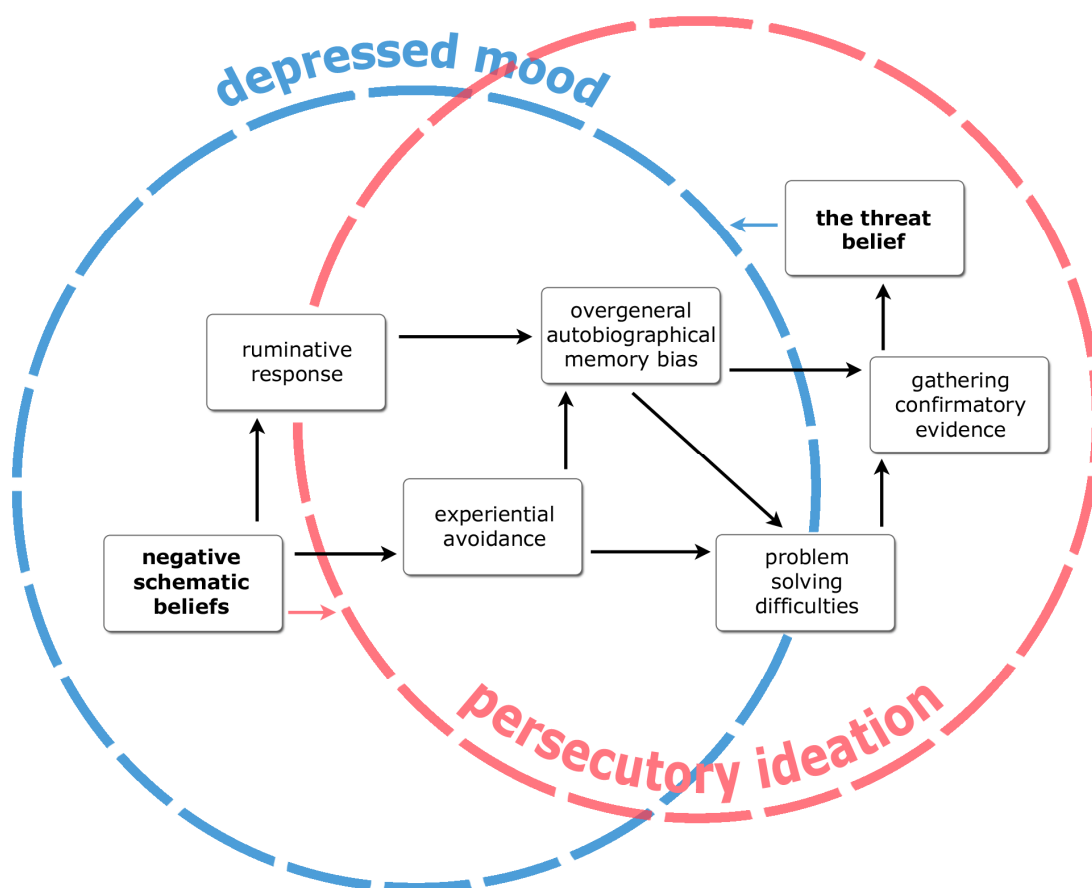


Figure 4.1. An integrated model of how depression-related cognitive factors might contribute to the persistence of persecutory delusions over time.

4.7 Summary

Compelling evidence exists that negative schematic beliefs predict the persistence of persecutory delusions over time, as they do with depression. In order to expand our understanding of the way that depressive cognitive factors might contribute to the maintenance of distressing persecutory beliefs, it will be helpful to apply the more detailed insights collected in the field of affective disorder to researching the cognitive maintenance mechanisms of paranoid beliefs.

Having reviewed the evidence connecting depressive cognition with delusions, a hybrid framework was put forward combining Williams' (2006) cognitive model of depression and Freeman et al.'s (2002) model of persecutory delusion persistence. As suggested by the broader scope of Freeman et al.'s original model, other factors than those reviewed here are also likely to contribute to the maintenance of threat beliefs. Some of these might be related to anxiety, which is very often concurrent with psychosis (Freeman & Garety, 1999) as well as with depression (Kaufman & Charney,

2000; Brady & Kendall, 1992). The novel empirical investigation of persecutory delusion persistence that is described in the following chapters will focus on cognitive processes related to depression, but measures of several other previously researched contributing factors will also be included, so that their relative effects can be observed within a single paradigm.

Chapter 5. Aims and methods of the present investigation

Findings linking depression with the persistence of persecutory delusions have supported the role of negative schematic beliefs in the maintenance of persecutory fears. Other depression related cognitive factors have scarcely been investigated in this population, and some findings have been difficult to interpret; for example, where the respective levels of depressive and psychotic symptoms in the studied participant groups were not systematically reported. The present investigation therefore sought to assess longitudinally the severity and persistence of persecutory delusions and of depression in a group of people with psychosis, and to examine the relationship between the cognitive factors identified in the previous chapter and the persistence of certain clinical symptoms. In order to allow comparisons to be made with findings from the depression literature, a group of participants with non-psychotic depression and a non-clinical control group were also included.

5.1 *Main aims*

1. To determine whether depression in people with persecutory delusions is associated with the same cognitive factors implicated in major depressive disorder: negative schematic beliefs, experiential avoidance, rumination, overly general autobiographical memory and problem solving difficulties.
2. To examine depression and depression-related cognitive factors as predictors of the persistence of persecutory delusions over time.

5.2 *Design*

Figure 5.1 summarises the experimental design. A group of people with persecutory delusions in the context of functional psychosis was recruited, and a comprehensive clinical interview assessment was used to determine which of these individuals would also meet diagnostic criteria for a current major depressive episode. This classification was used to split the group into two: a subgroup of people with delusions with concurrent depression, and a subgroup of people with delusions without concurrent depression.

In order to address the first aim of this investigation - to determine whether depression in people with persecutory delusions is associated with the same cognitive factors

implicated in major depressive disorder - the two subgroups of people with delusions (with and without depression) were asked to complete measures of schematic beliefs, experiential avoidance, rumination, autobiographical memory specificity and problem solving. The scores of the two subgroups were then compared, to examine which cognitive features might accompany depression specifically, when it occurs alongside persecutory delusions. In order that comparisons could be drawn with previously documented cognitive features of major depressive disorder, the same measures were administered to a group of people with non-psychotic depression and a non-clinical control group.

A prospective follow-up assessment was included to address the second aim of the project. All participants with delusions were re-assessed after six months (where possible), so that depression and associated cognitive factors could be tested as predictors of change in delusion severity over the follow-up period. Six months was chosen as a practically feasible follow-up period, intermediate in relation to other known longitudinal studies using similar participants and measures (Startup et al., 2007, three months; Green et al., 2008, six months; Fowler et al., 2011, 12 months; Drake et al., 2004, 18 months).

For the purposes of analysis, the cross-sectional and longitudinal components of the investigation will be treated separately, as Study One and Study Two.

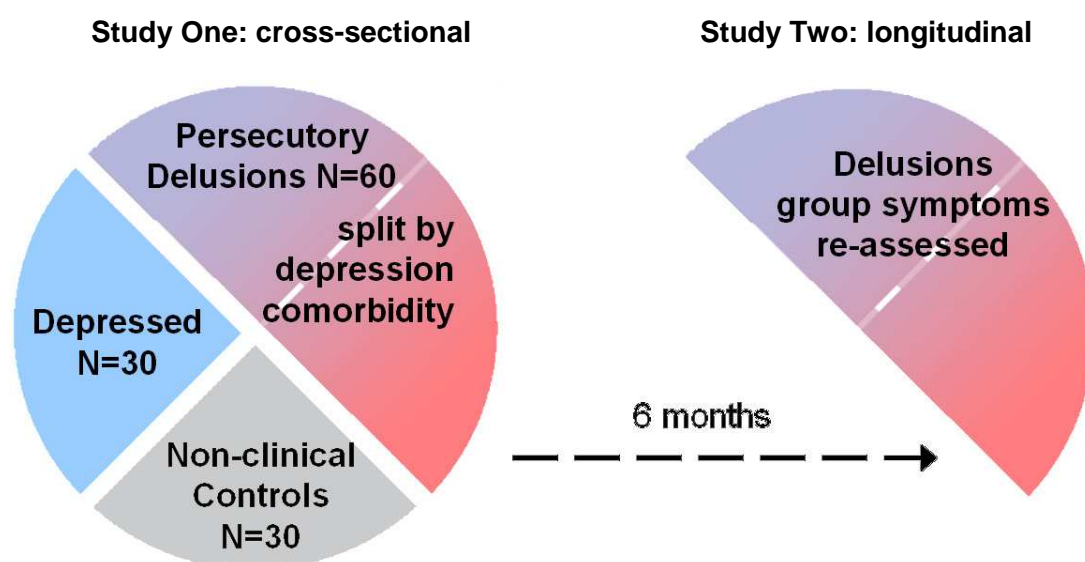


Figure 5.1. Design of the two linked studies.

5.3 Hypotheses

Each of the two studies had one main hypothesis, the evaluation of which was augmented by the testing of several secondary hypotheses. Secondary hypotheses concerned replicating previous findings and relating the results of the main analysis to them. Following the findings of previous research studies, it was expected that some cognitive factors would show specific associations with depression or with delusions, while other features would be elevated in all groups with significant psychopathology. The longitudinal analyses aimed to replicate the finding, observed in Chapter 2, that concurrent depression in groups with psychosis predicts the persistence of positive symptoms – focussing here explicitly on persecutory delusions – and to examine the possible cognitive mechanisms underlying this association.

5.3.1 Study One: cross-sectional

1. It was hypothesised that between 25% and 75% of participants with delusions would meet diagnostic criteria for major depression, so that the depressed and non-depressed subgroups would both be large enough for adequately powered comparisons to be made.

2. The primary hypothesis of Study One was that those among the delusions group who were found to be concurrently depressed, when compared to those who were not depressed, would show a pattern of cognitive features that has previously been linked with major depressive disorder:

- more negative schematic beliefs;
- more experiential avoidance;
- higher levels of rumination;
- less specific autobiographical memory;
- poorer problem solving performance.

In recognition of the continuous distribution of depression, the dimensional relationships between scores on these cognitive variables and depression severity were also examined within the combined delusions group.

The following secondary hypotheses related to additional analyses undertaken to contextualise the study's main findings in relation to the existing literature.

3. Depressed patients without delusions will show the same pattern of cognitive features described above when compared to non-clinical controls, replicating the findings of previous published studies.

4. Each of the clinical groups will show more negative schematic beliefs, higher levels of worry and poorer problem-solving performance than the non-clinical control group.

5. Participants with delusions will jump to conclusions more often than participants without delusions, irrespective of levels of depression.

The relationship between jumping to conclusions and problem-solving performance was also examined.

5.3.2 Study Two: longitudinal

1. The primary hypothesis of Study Two was that initial levels of depression and related cognitive factors would predict the persistence of persecutory delusions prospectively over six months.

2. It was also hypothesised that scores on depression-related cognitive factors would predict the persistence of depression after six months, in line with previous findings in the depression literature.

The relationships between changes in symptom severity and changes in cognitive process variables were also examined.

5.4 Ethical approval and research sites

An application was made through the Integrated Research Applications System for review of the project (comprising both the cross-sectional and longitudinal studies) by an NHS Research Ethics Committee and by the R&D department of each of the four London NHS Trusts where recruitment would take place: the South London and Maudsley NHS Foundation Trust, the Camden and Islington NHS Foundation Trust, the North East London NHS Foundation Trust and the Barnet, Enfield and Haringey Mental Health NHS Trust. The project was accepted into the NIHR portfolio. The Moorfields and Whittington NHS Research Ethics Committee reviewed the study and some amendments to the information sheets and consent forms were agreed. The committee declared a favourable opinion of the project (ref. 09/H0721/61). The R&D departments of the four NHS trusts also gave their approval for recruitment to go ahead.

A local collaborator was established at each site. Consultant psychiatrists, psychologists and team leaders of relevant community care teams were contacted to request their approval for recruitment of patients in their care, and presentations were made at team meetings to explain the project and to encourage referrals by nurses and social workers. Access was arranged to two registers of patients with an interest in research participation: the research register of the Social, Hope And Recovery Project (SHARP), in Lambeth, and the Psychological Intervention Clinic for outpatients with Psychosis (PICUP) research register, based in Southwark.

5.5 Participants

Based on the power calculations reported at the end of this chapter, 60 participants were recruited with persecutory delusions, 30 with non-psychotic depression and 30 non-clinical controls. Inclusion criteria are given for each group below.

5.5.1 Delusions group

Sixty patients were sought with current persecutory delusions as part of a schizophrenia-spectrum disorder: ICD-10 schizophrenia, delusional disorder or schizoaffective disorder. These inclusion criteria were chosen to mirror those used in other studies of persecutory delusions identified in the literature review, in order that the findings would be comparable.

The definition of a persecutory delusion used in this study was that given by Freeman & Garety (2000), who gave two main criteria that a delusion must meet to be classified as persecutory: that the individual believes that harm is occurring, or is going to occur, to him or her, and that the individual believes that the persecutor has the intention to cause harm.

Patients were recruited through inpatient and outpatient mental health services in four inner-city London NHS Trusts: the South London and Maudsley NHS Foundation Trust, the Camden and Islington NHS Foundation Trust, the North East London NHS Foundation Trust and the Barnet, Enfield and Haringey Mental Health NHS Trust.

Clinical care teams suggested the names of 197 people with psychosis for participation in the study, and the Participant Information Sheet was given to these individuals so that they could consider taking part. The experimenter spoke to 103 of these people, who had been happy for their contact details to be passed on. Of these 103, 21 were not interested in participation or were too busy, eight were excluded

because they did not hold current persecutory beliefs, eight said they were willing to take part but lost contact before an appointment could be arranged, three initially wanted to take part but then changed their minds, two did not attend arranged appointments and subsequently lost contact, and one completed half of the assessment measures and did not want to continue. Sixty people with delusions consented and completed the first assessment.

All of the study participants were outpatients at the time of assessment. 27 of the 60 participants were recruited through community mental health services in the Barnet, Enfield and Haringey Mental Health NHS Trust. 26 participants were recruited through the South London and Maudsley NHS Foundation Trust, of whom four were contacted through the SHARP research register and three through the PICUP research register. Seven participants were recruited through services in the Camden and Islington NHS Foundation Trust.

5.5.2 Depression group

30 people with major depression and without any history of psychosis or bipolar affective disorder were sought through outpatient services in the NHS trusts listed above. Some participants were referred to the study by clinical team members, and some referred themselves after seeing posters that were displayed in the teams' waiting rooms. Additionally, nine people who heard about the study through non-clinical recruitment were significantly depressed at the time of contact, and were therefore included in this group. The SCAN clinical interview schedules (see section 5.6.1) were used to confirm diagnoses and the absence of psychotic symptoms. All people with depression who were not already in contact with mental health services were given information about the ways that they could access support in their borough and online if they should like to do so.

Clinical teams suggested the names of 12 people for participation in the study, and the Participant Information Sheet was given to these individuals so that they could consider taking part. The experimenter spoke to eight of these people, of whom one refused to take part, one was excluded because she had auditory hallucinations, and one lost contact before an appointment could be arranged. Five of the people suggested by teams completed the assessments. All of these were referred by services in the South London and Maudsley NHS Foundation Trust, of whom two were contacted through the SHARP research register.

23 people contacted the study after seeing posters in waiting rooms. Two of these were excluded for having psychotic symptoms, four lost contact, one changed their mind about taking part and 16 completed the study assessments.

Of the nine depressed people recruited who came into contact with the study through the non-clinical recruitment route, five had been re-contacted after participating in other research (Freeman et al., 2008; Freeman, Pugh, Vorontsova, & Southgate, 2009; Freeman et al., 2010), and four had responded to posters seen around the local community. The advertisement asked for people with no history of mental health problems, and individuals were not sure whether the levels of difficulty that they experienced would disqualify them from participation. Participants joined the depressed group after the SCAN was used to confirm that they met diagnostic criteria for major depression. The severity of depression in this group, as measured by the BDI, was not significantly different from that in the group recruited through clinical services ($t(28) = -1.598, p = .12$).

5.5.3 Non-clinical control group

Non-clinical control participants with no history of psychosis or of depression were sought through two routes. 115 people were re-contacted after having taken part in previous studies (Freeman et al., 2008; Freeman et al., 2009; Freeman et al., 2010), for which they had volunteered after receiving a flyer in the post, and had declared an interest in further research participation. Additionally, leaflets were distributed in local community settings, including Camberwell Job Centre, Camberwell Library and local cafes.

Interest was expressed in the study by 63 people, and telephone contact was established with 53 of them. Nine of these people were currently depressed, and were recruited into the depression group as described above. Six people were excluded from the study for having had a history of mental health problems (but no current depression), and eight people lost contact. 30 people consented to participate and completed the study assessments. 12 of these had been re-contacted after taking part in other studies, and 18 had initiated contact after seeing a poster locally.

5.5.4 Common exclusion criteria for all groups

Individuals were excluded from participation if they had any organic brain disorder or primary substance abuse disorder, or if they were unable to give informed consent, or possessed an insufficient grasp of the English language to complete the measures.

5.6 Materials

5.6.1 Clinical symptom interviews

Schedules for Clinical Assessment in Neuropsychiatry (SCAN) version 2.1 (World Health Organisation, 1998).

The SCAN 2.1 is a system of semi-structured clinician rated interview schedules for psychiatric diagnosis, developed by the World Health Organisation. At the core of the SCAN 2.1 is the Present State Examination, version 10.2 (PSE-10.2). Part 1 relates to disorders of mood or neurosis, and Part 2 covers psychotic and cognitive processes. Items are scored according to the intensity, frequency and duration of individual symptoms or experiences, which are cross-examined using definitions given in the glossary that accompanies the manual. The system permits classification of both current and lifetime psychiatric disorders according to DSM-IV or ICD-10 criteria. The computerized version, IShell, allows input of ratings as they are made throughout the interview, which can then undergo processing with the appropriate algorithms to produce a set of diagnoses. The SCAN is traditionally used by experienced clinicians, but it has been shown that even less experienced trained interviewers can administer it reliably (Rijnders et al., 2000). The present experimenter completed formal training in the use of the SCAN over 9th – 13th November 2009 at the WHO-accredited training centre at Nottingham University.

In the current study, all sections of the SCAN 2.1 were included which are used in the assessment of schizophrenia spectrum disorders and of major depression. The diagnostic interviews were voice recorded for all participants who consented. The results of the SCAN interviews were used to verify participants' clinical diagnoses. The main purpose of the interview was to establish which of the participants with delusions would also meet diagnostic criteria for concurrent depression: this classification was then used to split the group into two for comparison. A randomly selected 15 of the 60 participants with delusions' depression interviews were re-rated by an independent assessor, blind to the other's ratings, to establish the reliability of the experimenter's diagnostic classifications. The second rater was also trained in use of the SCAN, and had wide experience in the assessment of people with psychopathology. The two raters produced the same diagnosis for all but one of these interviews: this participant was judged by the experimenter as meeting criteria for major depression, but the second assessor's ratings just missed the diagnostic threshold.

Psychotic SYmptoms RAtIng Scales (PSYRATS; Haddock et al., 1999). The PSYRATS is a 17-item instrument rating delusions and hallucinations. Each item is given a rating from 0 to 4. The instrument has demonstrated very good inter-rater reliability (intra-class correlation coefficients ranging from 0.79 to 1.0) and test-retest reliability (intra-class correlation coefficient 0.70). Only the 6-item Delusions Scale was used here. These items relate to delusional conviction, preoccupation (frequency and duration), distress (frequency and intensity) and functional impairment.

Four items were added to the PSYRATS Delusions Scale to enhance the resolution of the measurement of the main dimensions of interest and to explore the role of avoidance. Each was rated as a percentage from 0 to 100. The items were delusional conviction, delusional distress, the degree of effort used to avoid thinking about the belief, and the degree of success in avoiding thoughts about the belief.

5.6.2 Clinical symptom questionnaires

Green et al. Paranoid Thoughts Scale (GPTS; Green et al., 2008). The GPTS is a 32-item self-report measure of paranoid thoughts over the last month, split into two parts. Part A assesses ideas of reference (e.g. "People have been dropping hints for me") and Part B assesses ideas of persecution (e.g. "I was sure someone wanted to hurt me"). Each part contains subscales for conviction, preoccupation and distress related to paranoid thoughts. Items are rated on a 1-5 scale, with higher numbers indicating a greater level of paranoid thinking. This instrument was developed explicitly for consistency with Freeman & Garety's (2000) definition of a persecutory delusion, and with the content of diagnostic questions used in the SCAN interview. Good internal consistency was shown in both patients with persecutory delusions ($\alpha = .90$) and non-clinical controls ($\alpha = .95$). High test-retest reliability was also found (intra-class correlation coefficient = 0.87 over a two week interval). Convergent validity was supported by correlations with other questionnaire measures of paranoia and delusional ideation: the Paranoia Scale (Fenigstein & Venable, 1992) and the Peters et al. Delusions Inventory (Peters et al., 1999). An advantage of the GPTS is that it captures paranoia severity across the spectrum: people with delusions of persecution were found to score significantly more highly than non-clinical controls, but with overlap between the distributions, and widely ranging scores in each (32-149 in the non-clinical group; 32-160 in the delusions group). In those with persecutory delusions, subscale scores also showed appropriate associations with corresponding item scores on the PSYRATS interviewer-rated delusions scale (Haddock et al., 1999), and clinical change over six months from baseline assessment was captured

by changes in scores that correlated significantly with PSYRATS change scores (Green et al., 2008).

Beck Depression Inventory (BDI-II; Beck et al., 1996). The BDI is a 21-item self-report measure of the cognitive, affective and somatic symptoms of depression. It has shown good internal consistency ($\alpha = 0.84$), moderate-high test-retest reliability (intra-class correlation coefficients from 0.48 to 0.86) and good concurrent validity with clinician-rated measures of depression. When administered to groups of people with psychosis, the correlation of the BDI with the psychosis-specific Calgary Depression Scale for Schizophrenia (CDSS; Addington et al., 1993) has been reported as approaching unity: $r = .91$ (Birchwood et al., 2000).

Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI is a 21-item self-report measure of the cognitive and physiological symptoms of anxiety. Participants rate the items (e.g. "Heart pounding or racing"; "Fear of losing control") according to how severely they have been bothered by each experience over the previous week (0 = not at all bothered, 1 = mildly, 2 = moderately, 3 = severely). The scale has demonstrated high internal consistency ($\alpha = 0.92$) and test-retest reliability over a week ($r = 0.75$).

5.6.3 Cognitive process questionnaires

Brief Core Schema Scales (BCSS; Fowler et al., 2006). The BCSS contain 4 subscales of 6 items each scored on a 0-4 scale, which assess endorsement of beliefs in four categories: Positive Self, Negative Self, Positive Other and Negative Other. Higher scores indicate greater belief endorsement. These scales were developed with non-clinical and psychosis groups, where the subscales demonstrated high internal consistency (α between 0.78 - 0.88), good convergent and discriminant validity in relation to associated measures, and encouraging test-retest reliability over 3 weeks (r between 0.70 - 0.84).

Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). This 22-item questionnaire measures to what extent the participant generally responds to feeling sad, down or depressed by ruminating on his/her symptoms and the causes and consequences of these. Each item (e.g. "Think about a recent situation, wishing it had gone better") is rated on a 1-4 frequency scale, with higher scores indicating a greater degree of rumination when distressed (1 = almost never; 4 = almost always). The scale has exhibited high internal consistency ($\alpha = 0.89$) and high test-retest reliability over 5 months ($r = 0.80$).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ was developed to measure trait worry style, with an emphasis on clinically significant worry. Its 16 items refer to the generality of worry across situations (e.g. "I am always worrying about something"), the excessiveness of worry (e.g. "my worries overwhelm me"), and its uncontrollability (e.g. "I know I should not worry about things, but I just cannot help it"), which reflect the clinical characteristics of generalised anxiety disorder (Molina & Borkovec, 1994). Each item is rated on a 5-point scale (1-5). Items 1, 3, 8, 10 and 11 are reverse scored (e.g. item 8: "I find it easy to dismiss worrisome thoughts"). Summed total scores can range from 16 to 80, with higher scores indicating a greater tendency to worry. The scale has demonstrated high internal consistency in clinical and nonclinical samples, with α values between 0.86 - 0.95 (Van Rijsoort, Emmelkamp, & Vervaeke, 1999; Meyer et al., 1990; Molina & Borkovec, 1994; Brown, Antony, & Barlow, 1992). Good convergent and discriminant validity have also been shown with a large sample of anxiety disorder patients (Brown et al., 1992).

Avoidance and Action Questionnaire (AAQ; Hayes et al., 2004). This measure of experiential avoidance has 9 items (e.g. "if I could magically remove all the painful experiences I've had in my life, I would do so"), and the endorsement of each is rated on a 1-7 scale. Items 1, 4, 5 and 6 are reverse scored (e.g. item 5: "I'm not afraid of my feelings"). Summed total scores can then range from 9 - 63, with higher scores indicating greater experiential avoidance. The AAQ was designed to assess the tendency towards avoidant coping, independent of the specific content of the experiences that one currently avoids. Hayes et al. (2004) examined the scale's psychometric properties in a series of studies with over 2400 participants in total, reporting satisfactory internal consistency ($\alpha = 0.70$) and four-month test-retest reliability ($r = 0.64$).

5.6.4 Verbal cognitive tasks

Autobiographical Memory Test (AMT; Williams & Broadbent, 1986). The AMT is widely used to assess the specificity of episodic memories accessed by people with emotional disorders (Williams et al., 2007). This task requires participants to recall a specific autobiographical memory in response to each of a set of positive and negative cue words, presented orally and printed on cards. The instructions define an appropriate memory as "an event that lasted less than a day, and occurred at a particular time and place." Three practice cues are used at first to confirm that the

participant understands the instructions. Positive and negative cue words are then alternated, and prompts are used when a participant fails to produce a specific response, up to a 60 second time limit for each cue. Memories produced are coded as either specific (e.g. "watching a film at the cinema last Saturday"), overgeneral-categorical (e.g. "going to the cinema"), overgeneral-extended (e.g. "the first year of college"), semantic associate (not a memory, e.g. "that word reminds me of my sister") or no response. Research has mostly focused on the specificity of the *first* memory response given to each cue. The present protocol additionally recorded the specificity of the memories produced after further prompting, up to the 60s time limit.

Williams & Broadbent's original (1986) task used five positive and five negative cue words presented in a fixed order: *happy, sorry, safe, angry, interested, clumsy, successful, hurt, surprised* and *lonely*. In the present study, six positive and six negative words were used in each of two alternate forms for assessment at the two time points. The same number of cues was used in a previous study of people with persecutory delusions (Kaney et al., 1999), and a higher number of words increases the probability that parametric analyses may be used. Cue words for the present study were selected from a pool used by Brittlebank et al. (1993), with the four sets of six words approximately matched according to emotionality ratings and Kucera-Francis frequency (Kucera & Francis, 1967). Negative and positive words were alternated with negative first, in order to end the procedure with a positive memory. The sets of words chosen for the alternate forms were as follows.

Form A: *tired, friendly, weakness, pleasant, guilty, proud, ugly, lucky, sad, carefree, mistake, calm.*

Form B: *failure, happy, hurt, excited, bored, relieved, hopeless, peaceful, rejected, sunny, blame, hopeful.*

Means-Ends Problem-Solving procedure (MEPS; Platt & Spivack, 1975). This task was devised as a broad measure of interpersonal problem-solving cognition. Participants are presented with the beginning and the end of each of a set of stories. Each beginning describes the protagonist in a situation with a particular need (e.g. having just moved into a new neighbourhood and wanting to make friends locally), and each ending shows the need having been met (e.g. the protagonist has many good friends and feels at home in the neighbourhood). The task requires the participant to provide the middle of the story, in the form of a sequence of means leading from the initial situation to the desired endpoint. Responses are then scored

on the number of “relevant means” (problem solving steps) produced, and on the overall effectiveness of the given solution.

The specific administration and scoring procedure used in this study was based on that developed by Marx, Williams and Claridge (1992). The task instructions presented a clear problem solving set, asking participants to provide the *ideal* solution for each problem, and to describe it in very specific terms so that anybody could follow the plan of action. The experimenter read the stories to each participant. Participants followed the content on A4 cards, which had the beginning of the story printed on the top and the end on the bottom, with the middle part left blank to clarify visually that this section was to be filled in. Answers were taken down by the experimenter verbatim, and audio recorded where participants had consented. They were then scored on (1) the total number of distinct relevant problem solving steps produced and (2) the overall effectiveness of the solution from 1 to 7 (where 1 = not at all effective and 7 = extremely effective), following the guidance in Platt & Spivack’s original manual (Platt & Spivack, 1975). The responses of the delusions group were re-scored by an independent rater, a graduate psychologist blind to participants’ clinical status, so that inter-rater reliability could be calculated. Marx et al. (1992) found Pearson correlation coefficients between two raters of $r = .82$ for number of relevant means, and $r = .86$ for overall effectiveness. In the present study, the correlation coefficients between the two raters were calculated as $r = .88$ for number of relevant means and $r = .84$ for overall effectiveness, both $p < .01$.

Marx et al. (1992) used four of Platt & Spivack’s original ten scenarios. These and two more of the originals were used in the present study, selected so that different interpersonal life areas would be represented, and divided into two alternate forms (of three scenarios each) for the two assessment time-points. The stories were presented in a fixed order.

Beads task (Garety et al., 1991). This is a probabilistic reasoning task that has been used extensively with people with delusions (reviewed in Freeman, 2007; Garety & Freeman, 1999). Participants are invited to decide from which of two hidden jars coloured beads are being drawn. The jars contain beads of two colours mixed in reverse proportions, in this case 60 red beads and 40 white beads in one jar, 40 red beads and 60 white beads in the other. Beads are drawn one by one in a fixed order, and after each bead the participant can either decide on the source jar or request another piece of evidence (bead). The participants are informed that each bead is returned to the jar after it has been drawn, so that the proportions of beads always

remain the same. The key variable is the number of beads requested before a decision is made. If the participant does not choose to make a decision after 20 beads have been drawn, they are asked to decide at this point. The beads were drawn in the following order:

White, Red, Red, White, White, Red, White, White, White, Red, White, White, White, White, Red, Red, White, Red, Red, White.

In order to classify people who “jump to conclusions” in their data gathering style, the most common cut-off point used is two beads drawn or fewer (Garety et al., 2005). The first two beads in the above sequence contain one of each colour, and thus do not indicate a probability that one or other colour is dominant in the chosen jar. Decisions made after 2 beads or fewer were coded as JTC, and decisions after 3 beads or more as not JTC.

Wechsler Test of Adult Reading (WTAR; Ginsberg, 2003). This instrument was developed and had norms established alongside the widely-used Wechsler Adult Intelligence Scale - Third Edition (WAIS-III; Wechsler, 1997), for the purpose of estimating the pre-morbid IQ (intelligence before possible cognitive decline or brain injury) of individuals aged 16 – 89 years old. Participants are required to read aloud a list of 50 words with irregular pronunciations. The number of correct responses is used in combination with the participant's age to derive an estimate of pre-morbid intellectual functioning.

Verbal fluency, or the FAS test (Benton & Hamsher, 1976). Verbal fluency is considered a broad measure of executive control / working memory capacity. In Benton & Hamsher's task, participants were given 60 seconds on each of three trials to generate as many words as possible starting with the letter *F*, then *A*, then *S*, excluding proper nouns and repetitions of the same word stem with a different suffix. Verbal fluency was measured by the sum of valid words produced in the three trials. In the present study just one letter was used on each testing occasion, as has been considered sufficient by previous investigators (e.g. Dalgleish et al., 2007; Rosen & Engle, 1997). The letter *S* was used in the first assessment session, and the letter *A* was used in the second.

5.7 Procedure

Table 5.1 summarises the measures used at the first and second assessment points.

Measures at initial assessment	Repeated at follow-up
<i>Clinical symptom questionnaires</i>	
Q1 Green et al. Paranoid Thoughts Scale	Yes
Q2 Beck Depression Inventory	Yes
Q3 Beck Anxiety Inventory	No
<i>Clinical symptom interviews</i>	
• Schedules for Clinical Assessment in Neuropsychiatry	No
• Psychotic SYmptoms RATing Scales: Delusions Scale	Yes
<i>Cognitive process questionnaires</i>	
Q4 Brief Core Schema Scales	Yes
Q5 Ruminative Response Scale	Yes
Q6 Penn State Worry Questionnaire	Yes
Q7 Avoidance and Acceptance Questionnaire	Yes
<i>Verbal cognitive tasks</i>	
T1 Autobiographical Memory Test	Alternate form
T2 Means-Ends Problem-Solving procedure	Alternate form
T3 Beads task	No
T4 Wechsler Test of Adult Reading	No
T5 FAS verbal fluency test	Alternate form

Table 5.1. Measures completed by participants with delusions at the first and second assessment points.

The non-psychotic depressed participants and non-clinical controls completed the same measures, a single time, with the exception of the PSYRATS.

5.7.1 Study One: cross-sectional

At baseline, comprehensive assessments were made of persecutory delusions, depression and the cognitive factors of interest. Diagnoses and presence of persecutory delusions were confirmed using the interview Schedules for Clinical Assessment in Neuropsychiatry (SCAN) version 2.1 (World Health Organisation, 1998). The SCAN depression schedule was used to divide patients with delusions into two subgroups: those who met ICD-10 diagnostic criteria for concurrent depression alongside their psychotic disorder in one subgroup, and in the other subgroup those who did not meet criteria for a depression diagnosis. The Psychotic SYmptoms RATing Scales (PSYRATS; Haddock et al., 1999) were also used to assess the current multidimensional severity of delusions. Alongside these established interview measures, participants completed a number of questionnaires and cognitive tasks

designed to measure the cognitive factors that were hypothesised to distinguish between groups.

The groups of non-psychotic depressed people and non-clinical control participants completed the same assessments as the participants with delusions, with the exception of the PSYRATS.

Assessments took place either at the Institute of Psychiatry, King's College London, or at the participant's care team base, with the exception of four participants, who were seen at home, because they had particularly strong fears about going outside and travelling by public transport.

5.7.2 Study Two: longitudinal

Six months after the first assessments, the participants in the delusions group were followed up for a repeat assessment of the main clinical and cognitive variables of interest.

5.8 *Missing data*

There were high completion rates of the measures, with minimal missing data.

5.8.1 Study One: cross-sectional

At first assessment there was 0.06% missing questionnaire data in total, excluding whole missed questionnaires, or 0.37% missing data if whole missing questionnaires were included in the calculation.

5.8.1.1 *Whole measures*

One participant in the delusions group did not complete the BAI or PSWQ or AAQ, due to some difficulty with the English language. This individual's WTAR score was also discarded as invalid. One further person in the delusions group did not complete AAQ due to comprehension difficulty, and one more did not complete the Beads task because of time constraints. One non-clinical participant did not complete the WTAR due to poor eyesight (she had completed the questionnaires at home using special magnifying equipment), and for one of the participants with delusions the standardised WTAR score could not be computed because he had not wanted to give his age.

5.8.1.2 Individual items

On the PSYRATS (only administered to people in the delusions group), one person did not give a percentage figure for delusional distress. Another participant did not answer the "duration of preoccupation" item or the "conviction" item.

Two people with delusions and one non-clinical participant each omitted one item of the GPTS. Data for the BDI and BAI were complete (except for the person who did not complete the whole BAI, as mentioned above). Four people with delusions each omitted one item of the BCSS. One depressed person omitted one item of the RRS. Two depressed people each omitted one item of the PSWQ. One person with delusions omitted one item of the AAQ.

For the individual missing items of the GPTS, BCSS, RRS, PSWQ and AAQ, each omission was replaced with the participant's mean score from the other items on the scale (or subscale, in the case of the BCSS). This was done after the necessary items' scores had been reversed in the PSWQ and AAQ.

5.8.2 Study Two: longitudinal

Of the 60 participants in the delusions group, six did not complete the second assessment. Two of these no longer wanted to participate, two could not be contacted, and two wanted to participate but were not able to attend an appointment in the necessary time before the end of data collection. The participants who completed the second assessment did not differ significantly from those who did not in baseline depression (BDI: $t(58) = .92, p = .36$) or paranoia (GPTS: $t(58) = -.55, p = .59$) scores.

Among the 90% of participants who did complete the second assessment, there was a total of 0.07% missing questionnaire data, excluding whole missed questionnaires, or 0.70% missing data if whole missing questionnaires were included in the calculation.

5.8.2.1 Whole measures

Of the 54 participants who took part in the second assessment, one did not complete the PSWQ or AAQ due to English language difficulty, and two more did not complete the AAQ (one due to comprehension difficulty, one due to time constraints).

5.8.2.2 Individual items

On the PSYRATS, one participant did not give a figure for how successful they were at avoiding thinking about the belief, as they no longer believed it to be true and never thought about it. One more participant did not give a percentage figure for the intensity of delusional distress.

One participant omitted one item of the BCSS, one person omitted an item of the RRS, one person omitted one item of the PSWQ, and two people omitted one item each of the AAQ. These individual missing values were replaced with participants' scale or subscale means, as described above for Study One.

5.9 Analyses and power calculations

All analyses were carried out using SPSS version 19. Two-tailed tests were used, with significance level set at $\alpha = .05$.

5.9.1 Study One: cross-sectional

The distributions of the data from the four groups of participants were examined for significant violations of normality and for differences in key demographic characteristics. The data met requirements for planned parametric analyses to go ahead.

In order to avoid the increased probability of Type 1 error associated with multiple testing, an omnibus multivariate analysis of variance (MANOVA) was used with post-hoc pairwise comparisons to test most of the study hypotheses. When sample sizes are equal, as in the present study, analysis of variance is quite robust against violations of the assumptions of normality and of homogeneity of variance (Field, 2009, p. 360). A one-way independent samples MANOVA was run, with group as between-subjects factor, and post-hoc Tukey's Honestly Significant Difference tests were used to examine specific hypotheses.

The main hypothesis was tested by comparing the scores obtained by the two subgroups with delusions on the measures of schematic beliefs, autobiographical memory, experiential avoidance, rumination and problem solving.

The depressed group without delusions were compared to the non-clinical control group on the same measures.

Each of the clinical groups was compared against the non-clinical control group on scores of schematic belief endorsement, worry levels and problem solving performance.

Dichotomised Beads Task scores were compared among the groups using chi-squared tests to examine any associations of delusions and depression with JTC.

A dimensional analysis was used to further examine the main hypothesis, in recognition of the continuous distribution of depression and the loss of power associated with dichotomised group comparisons. Within the combined delusions group, correlation coefficients were calculated between participants' levels of depression and cognitive factor scores.

Finally, analysis of variance was used to examine any association between JTC and problem solving performance.

5.9.2 Power calculations for cross-sectional analyses

Williams et al. (2007) reported an average effect size of $d = 1.12$ in 11 studies comparing AMT performance of depressed patients to that of non-clinical controls. Iqbal et al. (2004) found a difference in overgenerality with effect size $d = 1.02$ between post-psychotically depressed patients and non-depressed individuals following a psychotic episode. Assuming, conservatively, a 3:1 ratio of group size, a two group t-test with a 0.05 two-tailed significance level would have 80% power to detect an effect size of 1.00 when the total delusion sample size is 44.

In a group of people who had recently experienced a psychotic relapse, Smith et al. (2006) found correlations between depression and subscales of the Brief Core Schema Scales of between .42 and .62. This corresponds to a moderate effect size. In order to have 80% power to detect a correlation of .40 with a .05 two-tailed significance level, a sample of 47 patients with delusions would be required.

5.9.3 Study Two: longitudinal

The main hypothesis was tested by investigating depression and associated cognitive factors as predictors of change in delusions. Partial correlations were computed between follow-up delusion severity and baseline scores on depression and each cognitive factor of interest, having controlled for baseline delusion severity. This would reveal any significant associations with prognosis. A multiple linear regression model

was then run with severity of delusions at follow-up as the dependent variable, severity at baseline as a covariate and the significant factors identified by correlation analysis as predictors. If a significant increase in r^2 resulted from addition to the model of these factors, they could be accepted as significant predictors.

The second hypothesis was tested as above, but with depression at follow-up as the dependent variable and baseline depression as covariate. Partial correlations were computed to reveal cognitive factors associated with change, and a multiple linear regression model was then run with these as predictor variables so that significant r^2 increases could be detected.

5.9.4 Power calculations for longitudinal analyses

Scarce data were available regarding predictors of change over time on the PSYRATS Delusions Scale. A partial correlation of moderate effect size $r = .30$ between predictor and outcome would correspond to an r^2 increase for the model of .09. For a multiple linear regression model that already includes one covariate (baseline severity) to have 80% power to detect at .05 two-tailed significance level an increase in r^2 of .09, the sample size required varies depending on the correlation between the covariate and dependent. Given a correlation of large effect size $r = .70$, $r^2 = .49$, the sample size required would be 39. If the correlation between covariate (baseline severity) and dependent (follow-up severity) were $r = .60$, the sample size required to detect the same increase in r^2 would be 51.

Published longitudinal studies of people with persecutory delusions suggest successful follow-up rates of around 80%. Startup et al. (2007), for example, reported 83% follow-up after three months. Successful six month follow-up of 80% of the sample of 60 participants in this study would have produced longitudinal data for 48 participants.

Delusions are often relatively stable. Only nine out of 25 patients in the Startup et al. study showed change over time. Absence of measurable change has a large impact on statistical power. The present study used a longer follow-up interval and a larger sample size in order to increase the probability of capturing change.

5.10 Summary

An investigation was designed to address two principal aims with two linked studies: the first was cross-sectional, and the second was longitudinal. The first aimed to

determine whether depression in people with delusions is accompanied by the same cognitive factors identified in the literature on major depressive disorder, and the second aimed to examine these cognitive factors as predictors of delusion persistence over time. Specific hypotheses were devised, and the appropriate measures and statistical procedures were selected for their testing. Groups of participants were recruited from the populations of interest, with sufficient sample sizes to give at least 80% power to detect at .05 two-tailed significance levels the predicted effect sizes. The study protocols were administered to the participants, data were collected and scored, and the planned statistical analyses were carried out. The next two chapters report the results of Study One and Study Two, respectively.

Chapter 6. Study One: cross-sectional

In this chapter, the results are reported of a cross-sectional investigation of depression and associated cognitive factors in a group of 60 people with persecutory delusions, 30 people with non-psychotic depression and 30 non-clinical controls. The main aim of this investigation was to determine whether depression in people with persecutory delusions is associated with the same cognitive factors implicated in major depressive disorder: negative schematic beliefs, experiential avoidance, rumination, overly general autobiographical memory and problem solving difficulties. First, the group with delusions were assessed and classified into two subgroups according to which individuals met diagnostic criteria for current major depression. All groups completed measures of cognitive variables, as detailed in the previous chapter. Group differences and correlations between cognitive variables and symptom levels were examined to test theory-driven hypotheses about the cognitive features that accompany delusions and depression. Additional exploratory analyses were applied to investigate the relationships between cognitive features previously linked with depression and those previously linked with delusions.

6.1 Results

6.1.1 Participant characteristics

The persecutory delusions group were all outpatients at the time of testing, and ranged in age from 20 to 65 years old, with a mean age of 41 years. Table 6.1 presents further demographic details. Among the 60 participants with delusions, 45 (75%) had a case-note diagnosis of schizophrenia, six (10%) had a diagnosis of schizoaffective disorder, one (2%) was diagnosed with delusional disorder and eight (13.3%) had uncertainty about which schizophrenia-spectrum diagnosis was most appropriate. Of the 58 people with delusions for whom medication details were retrieved, 52 (90%) were currently taking antipsychotic medication. Doses of these medications were converted to chlorpromazine equivalents, and classified into low doses (up to 200mg chlorpromazine equivalent), medium doses (201mg to 400mg chlorpromazine equivalent) and high doses (above 400mg chlorpromazine equivalent). 14 participants (24%) were on low doses of antipsychotics, 17 people (29%) were on medium doses, and 21 people (36%) were on high doses. 20 participants (33%) in the delusions group were also taking antidepressant medication.

The depressed group ranged from 23 to 69 years old, with a mean age of 43 years. The non-clinical controls were aged between 19 and 66, with a mean age of 40 years.

Further demographic information is given in Table 6.1. None of the participants in these two groups had ever taken antipsychotic medication, and the non-clinical controls were free from anything prescribed for mental health. Of the 30 people with depression, 13 (43%) were taking antidepressant medication at the time of testing.

The delusions group contained more people with non-white ethnicity, $\chi^2(1) = 13.75$, $p < .01$, more people who classified themselves as unemployed, $\chi^2(1) = 22.56$, $p < .01$, and fewer people with formal higher education qualifications (undergraduate degree or above), $\chi^2(1) = 13.89$, $p < .01$, than the participants without psychosis. The lower estimates of pre-morbid IQ derived from the WTAR for the delusions group compared to the groups without psychosis, $t(115) = 5.36$, $p < .01$, are consistent with their lower reported levels of educational attainment, and the mean group scores are similar to those reported in other studies of similar groups (e.g. Freeman, Pugh, Vorontsova, Antley, & Slater, 2010; Startup, Freeman, & Garety, 2007).

6.1.2 Diagnosis of depression in people with delusions

Hypothesis 1 was that between 25% and 75% of participants with persecutory delusions would meet diagnostic criteria for major depression, so that the depressed and non-depressed subgroups would both be large enough for adequately powered comparisons to be made.

Of the 60 participants with delusions who were interviewed using the SCAN, 30 (exactly 50%) were found to meet ICD-10 diagnostic criteria for a current episode of major depression. Hypothesis 1 was thus supported. The delusions group was split into two according to this diagnostic classification. The group with persecutory delusions without depression will hereafter be referred to as PD, the group with persecutory delusions and depression will be referred to as PD+D, the group with non-psychotic depression will be referred to as D, and the non-clinical control group will be referred to as NC. The demographic characteristics of the four groups of participants are summarised in Table 6.1.

The PD+D group were taking significantly higher doses of antipsychotic medication (chlorpromazine equivalent $M = 505\text{mg}$, 95% CI [373mg, 636mg]) than the PD group ($M = 297\text{mg}$, 95% CI [203mg, 390mg]), $t(56) = 2.61$, $p = .01$. The PD+D group were also significantly more likely to be taking antidepressants than the PD group: 17 of 30 (57%) in the PD+D group were on antidepressants compared to 3 of 28 (11% of those with available medication data) in the PD group, $\chi^2(1) = 13.54$, $p < .01$.

The groups were all similar in age and sex ratio. A one-way independent samples ANOVA with group as between subjects factor showed no main effect of group on age, $F(3, 115) = 0.42$, $p = .74$, and a chi-squared test indicated no significant sex differences between the groups, $\chi^2(3) = 3.04$, $p = .39$.

Group	PD (N=30)	PD+D (N=30)	D (N=30)	NC (N=30)
Age mean (SD)	40.1 (10.7)	42.8 (9.6)	42.5 (13.1)	40.4 (13.1)
<i>Sex</i>				
Male	19 (63%)	17 (56.7%)	14 (46.7%)	13 (43.3%)
Female	11 (36.7%)	13 (43.3%)	16 (53.3%)	17 (56.7%)
<i>Ethnicity</i>				
White	8 (26.7%)	15 (50.0%)	23 (76.7%)	20 (66.7%)
Black Caribbean	8 (26.7%)	6 (20.0%)	4 (13.3%)	0
Black African	7 (23.3%)	3 (10.0%)	1 (3.3%)	4 (13.3%)
Black other	2 (6.7%)	4 (13.3%)	1 (3.3%)	0
Indian	2 (6.7%)	0	0	2 (6.7%)
Pakistani	0	0	1 (3.3%)	0
Other	2 (6.7%)	2 (6.7%)	0	3 (10.0%)
Not given	1 (3.3%)	0	0	1 (3.3%)
<i>Marital status</i>				
Single	25 (83.3%)	25 (83.3%)	20 (66.7%)	18 (60.0%)
Married	1 (3.3%)	2 (6.7%)	4 (13.3%)	6 (20.0%)
Divorced / Separated	4 (13.3%)	3 (10.0%)	6 (20.0%)	5 (16.7%)
Not given	0	0	0	1 (3.3%)
<i>Highest education level</i>				
None	6 (20.0%)	3 (10.0%)	4 (13.3%)	0
GCSE	5 (16.7%)	10 (33.3%)	4 (13.3%)	4 (13.3%)
AS/A Level	5 (16.7%)	5 (16.7%)	5 (16.7%)	5 (16.7%)
Diploma / Foundation degree	5 (16.7%)	6 (20.0%)	2 (6.7%)	2 (6.7%)
Undergraduate Degree	5 (16.7%)	4 (13.3%)	8 (26.7%)	11 (36.7%)
Postgraduate diploma	3 (10%)	2 (6.7%)	7 (23.3%)	7 (23.3%)
Doctoral degree	0	0	0	1 (3.3%)
Not given	1 (3.3%)	0	0	0
<i>Occupation</i>				
Working full-time	0	0	6 (20.0%)	5 (16.7%)
Working part-time	4 (13.3%)	1 (3.3%)	3 (10.0%)	10 (33.3%)
Unemployed under 1 year	2 (6.7%)	1 (3.3%)	6 (20.0%)	2 (6.7%)
Unemployed over 1 year	18 (60.0%)	23 (76.7%)	8 (26.7%)	2 (6.7%)
Student full-time	0	0	0	1 (3.3%)
Student part-time	2 (6.7%)	1 (3.3%)	2 (6.7%)	3 (10.0%)
Volunteer	3 (10.0%)	2 (6.7%)	2 (6.7%)	3 (10.0%)
Homemaker	1 (3.3%)	1 (3.3%)	0	1 (3.3%)
Retired	0	1 (3.3%)	3 (10.0%)	3 (10.0%)
<i>Wechsler Test of Adult Reading (WTAR)</i>				
Standardized score mean (SD)	89.41 (18.79)	93.14 (17.55)	106.07 (15.20)	108.07 (11.19)

Table 6.1. Demographic characteristics of the participant groups.

6.1.3 Delusion severity in the subgroups

The content of the main persecutory belief of each of the participants with delusions is given in Appendix D. Figures 6.1 and 6.2 summarise the multidimensional severity of delusions reported by participants in the PD and PD+D groups, as measured by PSYRATS items with specific reference to the main persecutory belief.

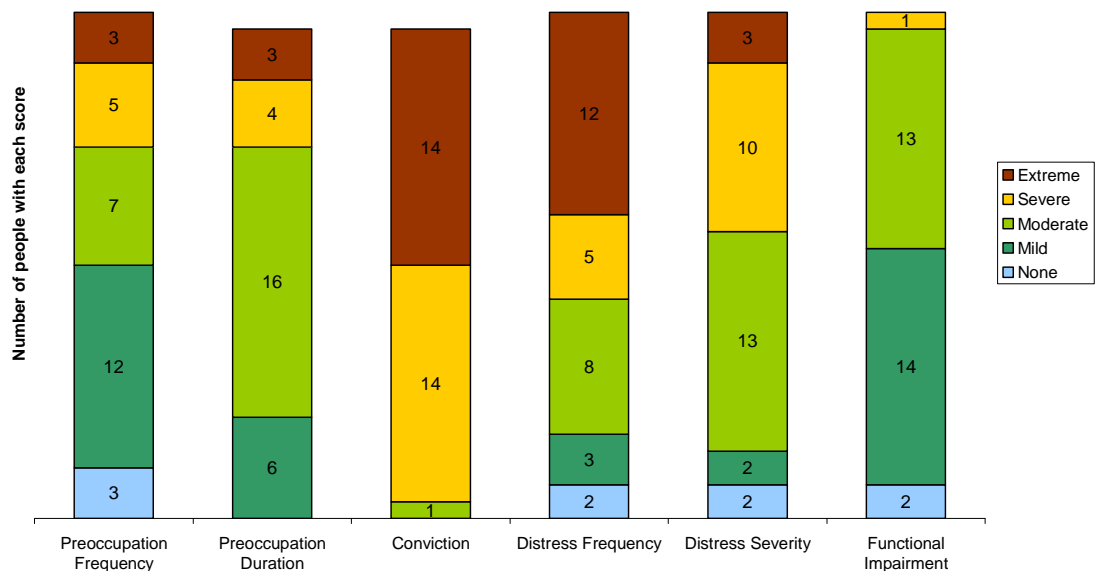


Figure 6.1. The multidimensional severity of persecutory delusions in the PD group.

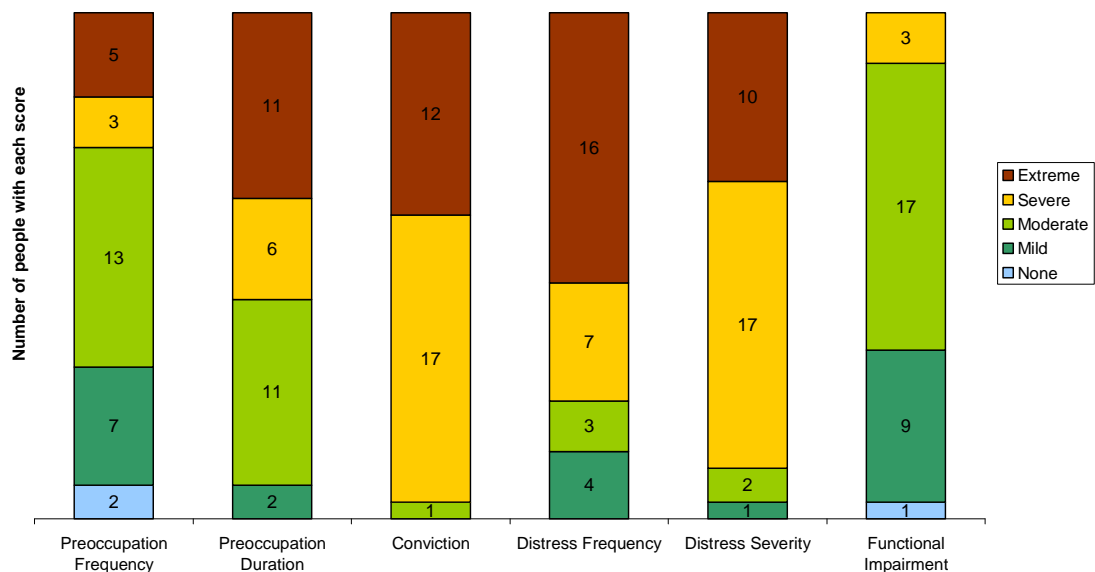


Figure 6.2. The multidimensional severity of persecutory delusions in the PD+D group.

The overall severity of persecutory delusions, as measured by the total score of the six 0-4 rated PSYRATS delusions scale items, was significantly higher in the PD+D group than the PD group, $t(57) = 2.77, p < .01$. The two groups' scores on the four supplementary percentage-scored items were compared to examine the multidimensional structure of the severity difference. No significant differences were found between the groups' reported conviction levels, $t(57) = 0.66, p = .51$, their efforts to avoid thinking about the content of the delusion, $t(58) = 1.20, p = .23$, or their success in avoiding such thoughts, $t(58) = 0.83, p = .41$. There was a significant difference in the amount of distress associated with the delusions, $t(57) = 3.29, p < .01$. The PD+D group reported being significantly more distressed by their persecutory beliefs ($M = 81.23\%$, 95% CI [74.39%, 88.08%]) than the PD group ($M = 60.79\%$, 95% CI [49.98%, 71.61%]).

6.1.4 Clinical symptom questionnaires and interviews

Table 6.2 summarises the four groups' scores on interview and questionnaire measures of clinical symptom severity.

Group	PD (N=30)	PD+D (N=30)	D (N=30)	NC (N=30)
PSYRATS mean total (SD)	13.76 (3.76)	16.40 (3.58)	n/a	n/a
GPTS mean total (SD)	94.40 (31.72)	110.50 (31.16)	65.70 (25.35)	41.54 (11.66)
SCAN				
Subclinical or no depression	30 (100%)	0	0	30 (100%)
Mild depression	0	5 (16.7%)	2 (6.7%)	0
Moderate depression	0	14 (46.7%)	12 (40%)	0
Severe depression	0	11 (36.7%)	16 (53.3%)	0
BDI				
Mean total (SD)	15.73 (7.87)	33.03 (14.86)	30.00 (8.96)	5.83 (4.00)
"Minimal" (0-13)	15 (50.0%)	1 (3.3%)	1 (3.3%)	30 (100%)
"Mild" (14-19)	6 (20.0%)	6 (20.0%)	2 (6.7%)	0
"Moderate" (20-28)	7 (23.3%)	6 (20.0%)	9 (30.0%)	0
"Severe" (29-63)	2 (6.7%)	17 (56.7%)	18 (60.0%)	0
BAI				
Mean total (SD)	14.97 (11.15)	29.48 (13.90)	22.20 (9.91)	4.70 (4.88)
"Minimal" (0-7)	10 (33.3%)	1 (3.3%)	2 (6.7%)	22 (73.3%)
"Mild" (8-15)	9 (30.0%)	5 (16.7%)	7 (23.3%)	7 (23.3%)
"Moderate" (16-25)	5 (16.7%)	4 (13.3%)	10 (33.3%)	1 (3.3%)
"Severe" (26-63)	6 (20.0%)	19 (63.3%)	11 (36.7%)	0
Missing	0	1 (3.3%)	0	0

Table 6.2. Clinical characteristics of the participant groups.

The relationships between clinical interview scores and questionnaire measure scores of symptom severity were examined. Within the combined group with delusions, a

significant correlation was found between PSYRATS total scores (relating to the current main persecutory belief) and GPTS total scores (relating to overall severity of persecutory ideation), $r = .38$, $p < .01$. The SCAN diagnostic algorithms classified depression levels as either none, mild, moderate or severe. A one-way independent samples ANOVA was run on the combined delusions group data, with SCAN depression severity as between subjects factor, to compare the BDI scores across the range of severity categories. A significant effect of group was found, $F(3, 56) = 20.55$, $p < .01$, with mean BDI rising incrementally with each SCAN depression severity step.

6.1.5 Comparison of cognitive factors across groups using MANOVA

Table 6.3 shows descriptive statistics of the four participant groups' scores on the main cognitive factors.

Group	PD (N=30*)	PD+D (N=30*)	D (N=30)	NC (N=30)
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>				
Self Negative	4.83 (3.57)	12.27 (7.06)	8.37 (4.57)	1.67 (1.49)
Self Positive	13.03 (5.94)	6.13 (5.32)	8.60 (4.90)	13.67 (4.85)
Other Negative	11.35 (6.83)	14.20 (6.85)	7.63 (4.57)	2.77 (4.19)
Other Positive	9.57 (5.79)	8.19 (6.96)	7.37 (3.95)	13.13 (4.47)
Avoidance (AAQ)	35.88 (7.79)	44.66 (7.04)	41.83 (6.80)	30.07 (6.45)
Rumination (RRS)	46.97 (12.11)	61.53 (13.90)	60.47 (9.32)	37.10 (9.73)
<i>Autobiographical Memory Task (AMT)</i>				
Total specificity	5.53 (3.06)	6.10 (2.82)	8.33 (2.38)	8.57 (2.08)
<i>Means-Ends Problem Solving (MEPS)</i>				
Total means	9.57 (4.39)	9.50 (3.79)	14.47 (5.02)	12.37 (3.36)
Effectiveness	10.27 (2.78)	10.20 (2.93)	13.50 (3.13)	13.03 (2.06)
Worry (PSWQ)	46.70 (11.83)	59.03 (12.37)	60.07 (10.72)	39.10 (13.03)

Table 6.3. Means and standard deviations (in parentheses) of the groups' cognitive factor scores.

* Two of the PD group did not complete the AAQ; one of the PD+D group did not complete the AAQ or the PSWQ.

The distributions of the groups' scores were examined to determine whether requirements for parametric testing would be met. For most measures, Shapiro-Wilk tests indicated that the groups' scores were normally distributed, skewness and kurtosis did not significantly differ from zero, and Levene's tests showed that the homogeneity of variance assumption was tenable. The distribution of the non-clinical control group's scores on the Negative Other subscale of the BCSS was the only one

with a kurtosis value significantly different from zero (kurtosis = 3.37, SE = 0.83, $z = 4.06$).

Analysis of variance is relatively robust against violations of the assumptions of normality and variance homogeneity when group sizes are equal, as they are here, so this method was deemed appropriate for the analysis. A multivariate one-way independent samples analysis of variance (MANOVA) was run, with group as the between subjects variable, to compare the groups' scores on the cognitive factors of interest. Using Pillai's trace, there was a significant effect of group on cognitive factor scores, $V = 1.10$, $F(30, 318) = 6.17$, $p < .01$. Univariate follow-up ANOVAs revealed significant main effects of group on every cognitive factor tested. Table 6.4 summarises these results. Post-hoc Tukey's Honestly Significant Difference tests were then used to test specific hypotheses. This procedure aims to keep the error rate below $\alpha = .05$, and has been recommended as the most favourable post-hoc method for power and robustness under homogeneous variance conditions (Stoline, 1981). Some of the mean scores quoted below in the context of pairwise comparisons differ from those given in Table 6.3, because cases were excluded in the MANOVA procedure: three participants with missing data were not included in the multivariate analysis.

Variable	Result
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>	
Self Negative	$F(3, 113) = 28.90$, $p < .01$
Self Positive	$F(3, 113) = 13.36$, $p < .01$
Other Negative	$F(3, 113) = 22.14$, $p < .01$
Other Positive	$F(3, 113) = 7.12$, $p < .01$
Avoidance (AAQ)	$F(3, 113) = 25.26$, $p < .01$
Rumination (RRS)	$F(3, 113) = 30.09$, $p < .01$
Memory specificity (AMT)	$F(3, 113) = 11.20$, $p < .01$
<i>Means-Ends Problem Solving (MEPS)</i>	
Total means	$F(3, 113) = 9.45$, $p < .01$
Effectiveness	$F(3, 113) = 12.06$, $p < .01$
Worry (PSWQ)	$F(3, 113) = 21.41$, $p < .01$

Table 6.4. Results of the one-way between subjects MANOVA.

6.1.6 Comparing the two groups with delusions

Hypothesis 2 - the main cross-sectional hypothesis of this study - was that the group with delusions and depression would show more negative schematic beliefs, more experiential avoidance, more rumination, less specific autobiographical memory and poorer problem solving than the group with delusions without depression.

Post-hoc Tukey's pairwise comparisons indicated significant differences between the PD group and the PD+D group's scores on negative beliefs about the self and positive beliefs about the self, both $p < .01$. There were no significant differences in negative beliefs about others, $p = .19$, or positive beliefs about others, $p = .68$. Figure 6.3 illustrates the BCSS subscale scores of the PD and PD+D groups.

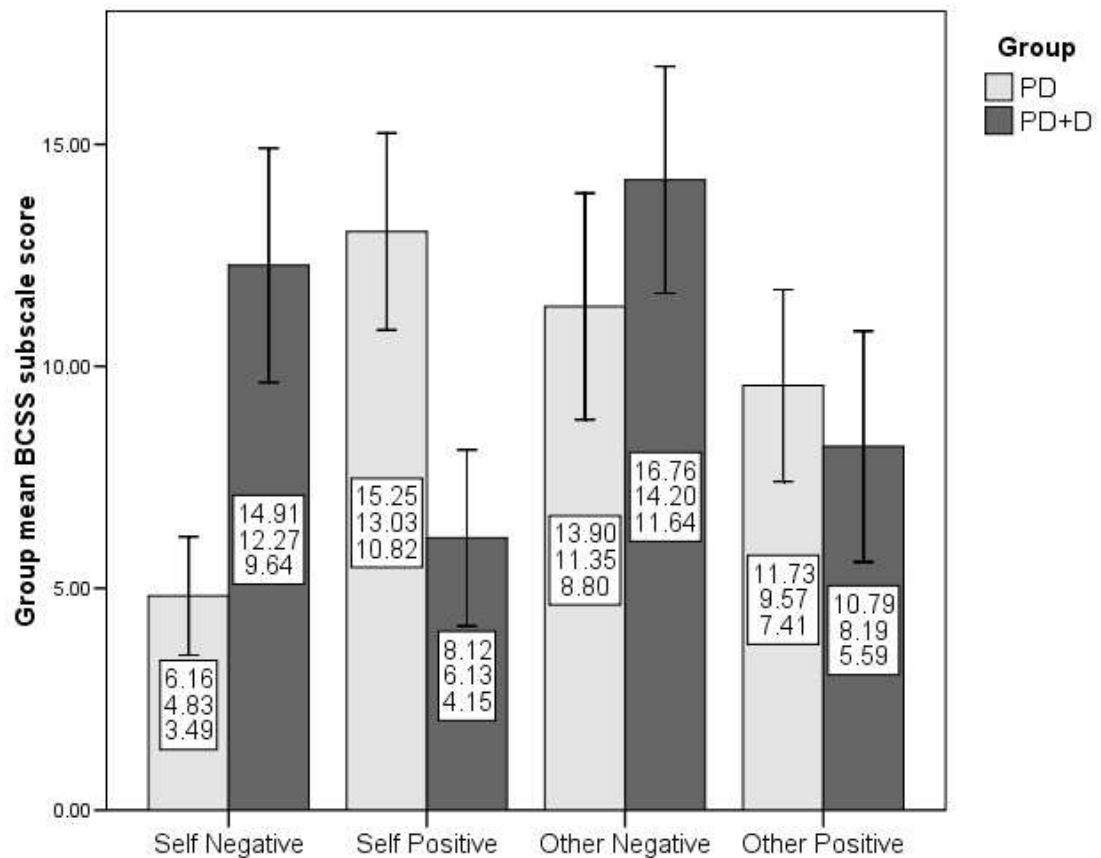


Figure 6.3. Brief Core Schema Scales (BCSS) subscale scores of the PD and PD+D groups.

Means are given between upper and lower bounds of 95% confidence intervals, which correspond to the error bars on the graph.

The effect sizes for the significant differences were calculated as Cohen's $d = 1.33$ for Self Negative beliefs and $d = 1.22$ for Self Positive beliefs, which both correspond to large effects (Cohen, 1992).

Because the BCSS subscale scores of the groups were not all normally distributed, Mann-Whitney U tests were also used to compare the scores of the two groups with delusions. The results were the same: the groups' scores differed on beliefs about the self, but not on beliefs about others.

Tukey's pairwise comparisons also indicated significant differences between the two delusions groups' experiential avoidance (AAQ) scores and their rumination (RRS) scores, both $p < .01$. The PD+D group's AAQ scores ($M = 44.66$, 95% CI [41.98, 47.33]) were significantly higher than those of the PD group ($M = 35.88$, 95% CI [32.86, 38.90]). The PD+D group's RRS scores ($M = 60.90$, 95% CI [55.69, 66.11]) were also significantly higher than those of the PD group ($M = 46.93$, 95% CI [42.08, 51.78]). The effect sizes for these differences were calculated as $d = 1.18$ for experiential avoidance scores and $d = 1.12$ for rumination, which both correspond to large effects.

No significant differences were indicated between the memory specificity (AMT) or problem solving (MEPS) scores of the two groups with delusions. Using the total number of specific first responses given to the 12 AMT cue words, the PD+D group's scores ($M = 6.03$, 95% CI [4.95, 7.12]) were not significantly different from the PD group's scores ($M = 5.39$, 95% CI [4.24, 6.55]), $p = .79$. This was the same regardless of whether scores for positive cues and negative cues were analysed separately or summed to a total. The analysis was repeated with a different scoring procedure: specific memories produced were counted up to the allotted 60 second limit, where previously only first responses had been counted. The pattern of results was the same. The MEPS total relevant means scores of the PD+D group ($M = 9.62$, 95% CI [8.18, 11.06]) were not significantly different from those of the PD group ($M = 9.61$, 95% CI [7.96, 11.25]), and the MEPS total effectiveness scores of the PD+D group ($M = 10.28$, 95% CI [9.15, 11.40]) were not significantly different from those of the PD group ($M = 10.25$, 95% CI [9.24, 11.26]), both $p > .99$.

Hypothesis 2 was partially supported. The group with depression alongside delusions reported more negative and less positive schematic beliefs about the self, more experiential avoidance and more rumination than the group with delusions without depression. However, the groups did not differ in terms of memory specificity or problem solving performance.

6.1.7 Comparing the two groups without delusions

Hypothesis 3 was that the group with non-psychotic depression would report more negative schematic beliefs, more experiential avoidance and more rumination than the non-clinical control group, and would show less specific autobiographical memory and poorer problem solving performance.

Post-hoc Tukey's pairwise comparisons indicated that the depressed group's scores differed significantly from those of the non-clinical controls, in the expected direction, on every schematic belief subscale of the BCSS, all $p < .01$. Figure 6.4 illustrates the BCSS subscale scores of the D and NC groups.

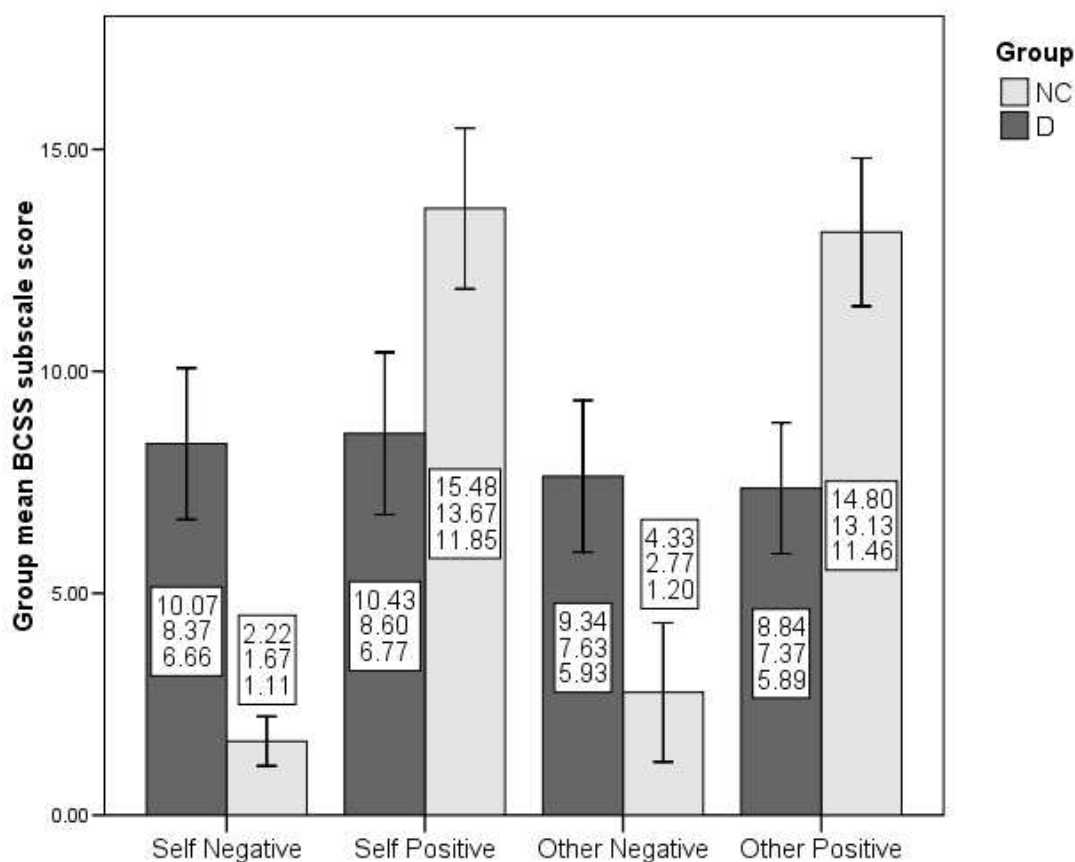


Figure 6.4. Brief Core Schema Scales (BCSS) subscale scores of the D and NC groups.

Means are given between upper and lower bounds of 95% confidence intervals, which correspond to the error bars on the graph.

The effect sizes of these differences were calculated as $d = 1.97$ for Self Negative scores, $d = 1.04$ for Self Positive scores, $d = 1.11$ for Other Negative scores and $d = 1.36$ for Other Positive scores, all corresponding to large effects.

Because the BCSS subscale scores of the groups were not all normally distributed, Mann-Whitney U tests were also used to compare the scores of the D and NC groups. The results were the same: the groups' scores differed on every BCSS subscale, all $p < .01$.

Tukey's tests also indicated significant differences between the D and NC groups' experiential avoidance (AAQ) and rumination (RRS) scores, both $p < .01$. The D

group's AAQ scores ($M = 41.83$, 95% CI [39.29, 44.37]) were significantly higher than those of the NC group ($M = 30.07$, 95% CI [27.66, 32.47]). The D group's RRS scores ($M = 60.47$, 95% CI [56.99, 63.95]) were also significantly higher than those of the NC group ($M = 37.10$, 95% CI [33.47, 40.73]). Effect sizes were calculated as $d = 1.77$ for AAQ scores and $d = 2.45$ for RRS scores, both corresponding to large effect sizes.

No significant differences were found between the scores obtained by the D group and the NC group on the memory specificity test (AMT) or the problem solving task (MEPS). Using the total number of specific first responses given to the 12 AMT cue words, the D group's scores ($M = 8.33$, 95% CI [7.44, 9.22]) were not significantly different from the NC group's scores ($M = 8.57$, 95% CI [7.79, 9.34]), $p = .99$. The analysis was repeated with positive and negative cues summed separately, and with total numbers of specific memories produced up to the 60 second time limit rather than just first responses, and in all cases the result was the same: no differences were indicated in performance between the two groups. The MEPS total relevant means scores of the D group ($M = 14.47$, 95% CI [12.59, 16.34]) were not significantly different from those of the NC group ($M = 12.37$, 95% CI [11.11, 13.62]), $p = .21$, and the MEPS effectiveness scores of the D group ($M = 13.50$, 95% CI [12.33, 14.67]) were not significantly different from those of the NC group ($M = 13.03$, 95% CI [12.26, 13.80]), $p = .91$.

Hypothesis 3 was partially supported. The group with non-psychotic depression reported more negative and less positive schematic beliefs about themselves and others, more experiential avoidance and more rumination than the non-clinical control group, but there were no differences found in memory specificity or problem solving.

6.1.8 Comparing each clinical group to the non-clinical control group

Hypothesis 4 was that each of the clinical groups would report more negative schematic beliefs and more worry than the non-clinical control group, and that the clinical groups would show poorer problem solving performance than the controls.

Tukey's tests indicated that the PD+D and D groups differed significantly from the control group, in the expected direction, on every schematic belief subscale of the BCSS, all $p < .01$. Negative Self scores of the PD+D group ($M = 12.21$, 95% CI [9.48, 14.95]) were significantly higher than those of the NC group ($M = 1.67$, 95% CI [1.11, 2.22]). Positive Self scores of the PD+D group ($M = 6.03$, 95% CI [3.99, 8.08]) were significantly lower than those of the NC group ($M = 13.67$, 95% CI [11.86, 15.48]).

Negative Other scores of the PD+D group ($M = 14.41$, 95% CI [11.80, 17.02]) were significantly higher than those of the NC group ($M = 2.77$, 95% CI [1.20, 4.33]). Positive Other scores of the PD+D group ($M = 7.92$, 95% CI [5.29, 10.56]) were significantly lower than those of the NC group ($M = 13.13$, 95% CI [11.46, 14.80]), all $p < .01$. Statistics regarding the differences between the D and NC groups' scores have already been reported in Section 6.1.7.

The PD group only differed significantly from the NC group on one of the BCSS subscales: Other Negative. The PD group's Other Negative scores ($M = 11.36$, 95% CI [8.60, 14.08]) were significantly higher than those of the NC group ($M = 2.77$, 95% CI [1.20, 4.33]), $p < .01$. The PD group's Other Positive scores ($M = 9.50$, 95% CI [7.36, 11.64]) were non-significantly lower than those of the NC group ($M = 13.13$, 95% CI [11.46, 14.80]), $p = .05$, and their Self Negative scores ($M = 4.71$, 95% CI [3.46, 5.96]) were non-significantly higher than those of the NC group ($M = 1.67$, 95% CI [1.11, 2.22]), $p = .06$. No significant difference was indicated between the PD group's Self Positive scores ($M = 12.64$, 95% CI [10.36, 14.92]) and those of the NC group ($M = 13.67$, 95% CI [11.86, 15.48]), $p = .88$.

Because not all of the groups' BCSS subscale scores were quite normally distributed, Mann-Whitney U tests were also used to compare each of the clinical groups to the non-clinical controls. Results of the tests indicated that each of the clinical groups differed significantly from the non-clinical controls on every BCSS subscale, except that the PD group did not differ from controls in positive beliefs about the self.

Pairwise comparisons of the groups' worry (PSWQ) scores indicated that both the PD+D group's scores ($M = 59.03$, 95% CI [54.33, 63.74]) and the D group's scores ($M = 60.07$, 95% CI [56.06, 64.07]) were significantly higher than those of the NC group ($M = 39.10$, 95% CI [34.23, 43.97]), both $p < .01$. The PD group's scores ($M = 46.04$, 95% CI [41.45, 50.62]) did not differ significantly from those of the NC group, $p = .13$.

Tukey's tests on the MEPS scores of the groups did not indicate any significant differences between the NC group and any of the other groups' total numbers of scored relevant means, though differences between the NC group and the PD and PD+D groups approached significance. The statistics regarding non-significant differences between the D and NC groups were given in the previous section. A non-significant trend was indicated towards MEPS total relevant means scores of the NC group ($M = 12.37$, 95% CI [11.11, 13.62]) being higher than the total relevant means

scores of the PD group ($M = 9.61$, 95% CI [7.96, 11.25]), $p = .06$, and the PD+D group ($M = 9.62$, 95% CI [8.18, 11.06]), $p = .06$.

Significant differences were indicated between the MEPS effectiveness scores of the NC group and the PD and PD+D groups, but not between the NC group and the D group. The MEPS effectiveness scores of the NC group ($M = 13.03$, 95% CI [12.26, 13.80]) were significantly higher than those of both the PD group ($M = 10.25$, 95% CI [9.24, 11.26]) and the PD+D group ($M = 10.28$, 95% CI [9.15, 11.40]), both $p < .01$.

Hypothesis 4 was partially supported. The clinical groups all held more negative beliefs about other people than controls, but only the two depressed groups held significantly more negative beliefs about themselves. The two depressed groups also scored more highly on worry than controls, but the PD group did not. The two groups with delusions were found to have significantly lower effectiveness scores on the problem solving task (MEPS) than controls, but none of the groups differed significantly from controls with regard to the total number of means scored.

6.1.9 Unexpected findings regarding memory specificity and problem solving performance

Tukey's pairwise comparisons following the MANOVA indicated that the memory performance of each of the two groups with delusions differed significantly from the performance of each of the two groups without delusions, regardless of levels of depression. The total specificity scores of the PD group ($M = 5.39$, 95% CI [4.24, 6.55]) and the PD+D group ($M = 6.03$, 95% CI [4.95, 7.12]) were lower than those of the D group ($M = 8.33$, 95% CI [7.44, 9.22]) and the NC group ($M = 8.57$, 95% CI [7.79, 9.34]), all $p < .01$. This pattern is similar to the findings given above for problem solving performance: the PD and PD+D groups scored significantly lower than the D and NC groups. This unexpected result was investigated further.

Both AMT and problem solving performance have been linked with executive capacity (Williams, 2006; Dalgleish et al., 2007), so associations were examined with verbal fluency scores. It has previously been reported that people with psychosis have reduced verbal fluency compared to non-clinical controls (e.g. Frith, 2004). The participants with delusions in the present study scored significantly lower on the verbal fluency task than those without delusions, $t(118) = 3.85$, $p < .01$. Within the combined delusions group, verbal fluency was significantly correlated with MEPS total means ($r = .40$, $p < .01$) and MEPS effectiveness scores ($r = .44$, $p < .01$), and the correlation between verbal fluency and AMT specificity approached significance ($r = .23$, $p = .07$).

Premorbid IQ estimates derived from the WTAR were significantly lower for participants with delusions than for those without delusions, $t(115) = 5.37, p < .01$. Within the combined delusions group, estimated IQ was significantly correlated with MEPS total means ($r = .35, p < .01$), MEPS effectiveness scores ($r = .28, p = .03$) and AMT specificity ($r = .38, p < .01$).

One-way independent samples ANOVAs with the presence of delusions as between-subjects factor and AMT and MEPS scores as dependent factors indicated a significant effect of delusions on AMT specificity, $F(1, 118) = 30.77, p < .01$, MEPS total means, $F(1, 118) = 25.43, p < .01$, and MEPS effectiveness, $F(1, 118) = 36.91, p < .01$. Adding verbal fluency as a covariate to these models did not account for the group effects: the presence of delusions still had a significant main effect on each of the AMT and MEPS scores, all $p < .01$. Similarly, when WTAR scores were added (instead of verbal fluency) as a covariate, the effects of delusion presence on the AMT and MEPS scores all remained significant, all $p < .01$.

6.1.10 Jumping to conclusions

Hypothesis 5 was that more of the participants with delusions than of those without delusions would jump to conclusions (JTC) on the Beads Task, irrespective of levels of depression.

The JTC data were dichotomised, and a chi-square test was used to compare the numbers of people who did and did not JTC in the combined delusions group to the corresponding numbers in the combined group without delusions. Of the 59 people with delusions who completed the Beads Task, 25 jumped to conclusions (where JTC is defined as making a decision after two beads or fewer). Of the 60 people without delusions, 5 jumped to conclusions. There was a significant association between the presence of delusions and JTC, $\chi^2(1) = 18.28, p < .01$, such that people with delusions were significantly more likely to jump to conclusions than those without delusions.

To examine any association of depression with JTC, chi-square tests were used to compare the numbers of people who did and did not JTC in the subgroups with and without depression within each delusion status group. Of the 29 people in the PD group who completed the Beads Task, 18 jumped to conclusions. Of the 30 people in the PD+D group, 7 jumped to conclusions. In the D group, 3 of 30 JTC, and in the NC group, 2 of 30 JTC. Within the delusions group, there was a significant association

between the presence of depression and JTC, $\chi^2(1) = 9.06$, $p < .01$, such that people with delusions and depression were significantly less likely to jump to conclusions than people with delusions without depression. No association of depression with JTC was found in the groups without delusions.

Hypothesis 5 was supported by the finding that participants with delusions were significantly more likely to JTC than those without delusions. However, the prediction that JTC rates would be unrelated to depression was contradicted by the finding that among participants with delusions, those who were concurrently depressed were significantly less likely to JTC than those who were not concurrently depressed.

The unexpected finding of less JTC in the PD+D group than the PD group was explored further. The mean number of beads drawn to decision by the PD+D group ($M = 4.87$, 95% CI [3.26, 6.47]) was very close to that of the PD group ($M = 4.86$, 95% CI [2.65, 7.07]), $t(57) < .01$, $p < .99$. Cross-tabulation of the numbers of beads drawn by individuals in the groups revealed that 13 people in the PD group and 7 in the PD+D group decided after one bead, 5 people in the PD group and none in the PD+D group decided after two beads, and 1 person in the PD group and 11 people in the PD+D group decided after 3 beads. Thus, if scores had been dichotomised at 3 beads or fewer for JTC rather than 2, the two groups with delusions would not have appeared significantly different. Of the PD group, 19 people had decided by 3 beads, and in the PD+D group, 18 people had decided, $\chi^2(1) = 0.19$, $p = .66$.

6.1.11 Dimensional analysis of the combined delusions group

Correlation analysis was used to investigate the associations between levels of depression and cognitive factor scores in the combined delusions group. The Beck Depression Inventory (BDI) was used as the dimensional measure of depression for these analyses. The distribution of BDI scores in the combined delusions group showed significant positive skew (skewness = 0.84, SE = 0.31, $z = 2.71$), so Spearman's rank correlation coefficients were calculated as measures of association.

Table 6.5 summarises Spearman's r_s coefficients and associated significance values of the correlations between BDI depression levels and cognitive factor scores. The results indicated significant positive correlations between BDI depression levels and schematic beliefs, experiential avoidance, rumination and worry. Correlations of large effect size (Cohen, 1992) were found between higher levels of depression and more negative beliefs about the self, fewer positive beliefs about the self, more experiential avoidance, more rumination and more worry. Depression did not show any significant

association with memory specificity or with problem solving performance. Memory specificity and problem solving performance scores were correlated with each other.

	BDI	SN	SP	ON	OP	AAQ	RRS	AMT	MEPS	
									M	E
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>										
Self Negative	.75** < .01									
Self Positive	-.67** < .01	-.52** < .01								
Other Negative	.24 .07	.49** < .01	-.05 .68							
Other Positive	-.26* .04	-.18 .16	.40** < .01	-.06 .68						
Avoidance (AAQ)	.64** < .01	.53** < .01	-.59** < .01	.26 .05	-.28* .04					
Rumination (RRS)	.62** < .01	.51** < .01	-.28* .03	.31* .02	.05 .69	.46** < .01				
<i>Autobiographical Memory Task (AMT)</i>										
Total specificity	-.05 .71	.03 .80	-.07 .58	-.24 .06	-.05 .72	.04 .75	-.02 .90			
<i>Means-Ends Problem Solving (MEPS)</i>										
Total means	.04 .77	.03 .85	-.12 .35	-.10 .47	-.27* .04	.08 .55	.14 .28	.33* .01		
Effectiveness	-.04 .77	-.08 .55	-.03 .82	-.13 .31	-.22 .10	.08 .54	.11 .40	.34** < .01	.92** < .01	
Worry (PSWQ)	.59** < .01	.49** < .01	-.47** < .01	.15 .26	-.19 .14	.59** < .01	.44** < .01	.06 .65	.07 .60	.08 .57

Table 6.5. Spearman's rank correlation coefficients between depression and cognitive factor scores in the combined delusions group.

Significance values are given below coefficients. ** indicates significance at $p < .01$ level; * indicates significance at $p < .05$ level.

6.1.12 Jumping to conclusions and problem solving in people with delusions

A one-way independent samples MANOVA was run on the combined delusions group data to compare the MEPS total means and effectiveness scores of those who jumped to conclusions on the Beads Task and those who did not JTC. Using Pillai's trace, the multivariate effect of JTC on MEPS scores just missed significance, $V = 0.10$, $F(2, 56) = 3.15$, $p = .051$. The high correlation ($r_s = .92$) between the two MEPS measures may have caused the MANOVA to lose power, a phenomenon reported by Ramsey (1982). Univariate follow-up ANOVAs indicated significant effects of JTC on both the total relevant means scores ($F(1, 57) = 6.40$, $p = .01$) and effectiveness scores ($F(1, 57) = 5.15$, $p = .03$). Inspection of the descriptive statistics indicated that

the 25 people with delusions who did JTC scored fewer MEPS means ($M = 8.20$, 95% CI [6.57, 9.83]), than the 34 who did not JTC ($M = 10.74$, 95% CI [9.44, 12.03]), and that those who JTC also scored lower on effectiveness ($M = 9.40$, 95% CI [8.32, 10.48]) than those who did not JTC ($M = 11.00$, 95% CI [10.05, 11.95]).

Those among the combined delusions group who JTC had significantly lower WTAR scores ($M = 83.92$, 95% CI [76.41, 91.43]) than those who did not JTC ($M = 96.72$, 95% CI [90.79, 102.64]), $t(55) = 3.01$, $p < .01$. Adding WTAR scores as a covariate to the ANOVA models above reduced the main effects of JTC. With MEPS total relevant means as dependent factor, the main effect of JTC was no longer significant, $F(1,54) = 3.18$, $p = .08$, and the same was the case with MEPS effectiveness scores as the dependent variable: the main effect of JTC missed significance, $F(1,54) = 2.88$, $p = .10$.

6.2 Discussion

The finding that exactly half of the participants with persecutory delusions currently met ICD-10 research diagnostic criteria for major depressive disorder is consistent with reports of other studies in psychosis (Birchwood, Iqbal, & Upthegrove, 2005; Buckley et al., 2009), and indicates substantial prevalence of clinically significant depression in this group. Over half (57%) of the people with delusions who met diagnostic criteria for depression were prescribed antidepressant medications, which suggests recognition by clinical teams of depression as a significant symptom dimension for treatment in this group. The subgroup with concurrent depression experienced delusions of higher severity overall than those who were not depressed, and this difference was marked in the levels of distress associated with persecutory beliefs, rather than the strength of their conviction.

6.2.1 Cognitive factors associated with depression

The primary cross-sectional hypothesis was that those among the delusions group who were found to be concurrently depressed, when compared with those who were not depressed, would show a pattern of cognitive features that has previously been linked with major depressive disorder:

- more negative schematic beliefs;
- more experiential avoidance;
- higher levels of rumination;
- less specific autobiographical memory;
- poorer problem solving performance.

Some, but not all, of the hypothesised depression-related cognitive features were elevated in the PD+D group compared to the PD group. The results showed that those with delusions who were also depressed had more negative schematic beliefs about themselves and reported more experiential avoidance (EA) and more rumination than those with delusions who were not concurrently depressed. However, no significant differences were evident in schematic beliefs about others, memory specificity or problem solving performance.

In order to contextualise this pattern of group differences in relation to previous reports about the features of major depressive disorder, the same comparisons were made between a group of people with non-psychotic depression and a group of non-clinical controls. The pattern of results was the same as that between the two groups with delusions, with the additional difference that the depressed group had more negative schematic beliefs about other people than the non-clinical control group. Again, no evidence was found for a difference in memory specificity or problem solving performance.

Dimensional analysis of the associations between depression levels and cognitive variables in the combined delusions group supported the associations found of depression with schematic beliefs, EA and rumination. It was additionally found that worry was associated with high levels of depression. This last finding was not related to any *a priori* hypotheses, and might be a characteristic of the present sample rather than the wider population of interest.

6.2.1.1 Schematic beliefs

The hypothesised elevation of negative schematic beliefs in people with persecutory delusions and depression compared to those without depression was confirmed in this group. The findings indicated that depression occurring alongside delusions was accompanied by more negative schematic beliefs, particularly about the self. It is perhaps unsurprising that no significant differences were found in beliefs about other people, which were very negative in both of these subgroups, in line with the common presence of persecutory delusions. Comparisons of the schematic belief scores of the group with non-psychotic depression and the non-clinical controls confirmed the expectation that the depressed group endorsed more negative and fewer positive beliefs than controls, in relation to the self and others.

The finding that the PD group without depression only differed significantly from controls on one BCSS subscale – Other Negative, as fits with the presence of persecutory delusions – while the PD+D group scored substantially more negative and less positive beliefs than controls on all subscales, is consistent with reports in the literature of wide variation in the self esteem of people with persecutory delusions, and suggests that part of this variation may be associated with different levels of concurrent depression. This is not to say that the PD group's scores on all three other schematic belief subscales were very similar to those of the NC group. The differences between Self Negative and Other Positive subscale scores of the PD and NC groups approached significance (both reached significance if a Mann-Whitney test was used instead of Tukey's HSD), while the Self Positive subscale scores of the two groups did not show any indication of a significant difference.

6.2.1.2 Responses to distress: experiential avoidance and rumination

The pattern of responding to unpleasant phenomena with avoidance or rumination that has been reported previously in people with depression was replicated in this study in people whose depression accompanied persecutory delusions. People with persecutory delusions and concurrent depression reported significantly more experiential avoidance and more of a tendency to ruminate in response to distress than those with delusions without depression. Levels of EA and rumination similarly distinguished a group of non-psychotic depressed people from non-clinical controls, as predicted. The effect sizes of these differences were all comparably large. Dimensional analysis of the combined delusions group indicated that levels of EA and rumination were strongly correlated with severity of depression.

Experiential avoidance and ruminative response style have been conceptualised in the depression literature as relatively stable trait characteristics, which predispose individuals to respond to distress in certain ways that may be unhelpful and can lead to the perpetuation of emotional disorder. An alternative proposal would be that questionnaires such as the AAQ and RRS measure mood-dependent cognitive phenomena that are symptoms, rather than causes, of depression. It would not be warranted to devote a great deal of attention to phenomena that are causally posterior and unimportant in determining subsequent prognosis of principal clinical variables (such as distress and occupational functioning). The relevance of researching processes such as experiential avoidance and rumination rests on the premise that they affect subsequent clinical symptoms, such as low mood. Observational association studies, such as that described in this chapter, cannot discriminate between causal frameworks of opposite direction. However, prospective studies have

found that ruminative response style can predict the onset, severity and duration of depressive episodes (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 1994; Spasojević & Alloy, 2001), and manipulation studies have supported a causal effect of these response patterns on subsequent mood states. Depressed people induced experimentally to ruminate show worsening of low mood (Nolen-Hoeksema & Morrow, 1993; Donaldson & Lam, 2004). Similarly, experimental induction of a conceptual-evaluative form of self-focussed attention (another description of the pathological ruminative mindset) has been shown to cause low mood in those with high trait ruminative response style (Watkins, 2004). With regard to the processes that experiential avoidance invokes, both thought suppression (cognitive avoidance) and emotional suppression (emotional avoidance) induced experimentally in relation to unpleasant content have been found to result in subsequent increased distress (e.g. Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Marcks & Woods, 2005). Rumination-focused CBT and experiential avoidance-focused Acceptance and Commitment Therapy have both demonstrated clinical efficacy in treating depression (Watkins et al., 2007; Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Zettle & Rains, 1989). It is true that rumination and avoidance occur in response to distress, and in this way are a symptom of it, but the pattern of findings here summarised suggests that the degree of inclination towards these particular distress responses can have an effect on the degree or duration of subsequent affective disturbance experienced. Returning to the findings of the present study, it would be premature to suggest that depression in the PD+D group was caused by tendencies towards rumination or avoidance, but it is possible that these response tendencies might be associated with symptom persistence in this group with delusions and depression in an analogous way to that reported in major depressive disorder. Longitudinal investigation will be necessary to examine this possibility further.

Worry – a construct that shares with rumination the form of negative, repetitive thinking but is more future-orientated - was also found to correlate strongly with levels of depression in the combined delusions group. It has previously been found that worry can predict the persistence of delusions over time (Startup et al., 2007) and that treating worry in psychological therapy can reduce the distress associated with persecutory delusions (Foster et al., 2010). The results of the present study suggest that those people with delusions who have high levels of worry might also have high levels of depression. However, this was an exploratory finding of the present study, with no corresponding *a priori* hypothesis, and as such cannot be said to generalize to the population beyond the present sample.

6.2.2 Unexpected findings regarding memory specificity and problem solving performance

No evidence was found for the predicted associations of depression with overgeneral memory bias (OGM) and problem solving difficulties. Not only were these features not found the subgroup with delusions and depression compared to those with delusions without depression, but there was also no difference in either of these performance scores between the non-psychotic depressed group and non-clinical controls.

Significant differences were found, in both memory and problem solving performance, between the groups with delusions and the groups without delusions. Thus the unexpected findings have two principal aspects: the non-replication of previously reported associations of task performance with depression, and the finding instead of significantly reduced performance by participants with delusions compared to those without delusions.

6.2.2.1 Non-replication of association of depression with AMT and MEPS task performance

The non-replication here of an overgeneral autobiographical memory bias (Williams et al., 2007) and problem-solving deficits (Marx et al., 1992; Watkins & Baracaia, 2002) that have previously been found by a number of studies in people with depression might be explained by differences between the participant samples used in comparison groups. Efforts were made in the present study to recruit community control participants who would be demographically comparable to the patient groups, and as a result the non-clinical control group might have been more similar to the others than some used in previous studies. Table 6.6 summarises the four groups' average item scores on the MEPS from the present study (Table 6.3 reported total scores summed from three items), alongside the corresponding figures given by Marx et al. (1992).

Group	MEPS average means	(SD)	MEPS average effectiveness	(SD)
<i>Present study</i>				
PD (N=30)	3.19	(1.46)	3.42	(0.93)
PD+D (N=30)	3.16	(1.26)	3.40	(0.98)
D (N=30)	4.82	(1.67)	4.50	(1.04)
NC (N=30)	4.12	(1.12)	4.34	(0.69)
<i>Marx et al. (1992)</i>				
D (N=20)	6.75	(6.08)	3.71	(1.39)
NC (N=20)	11.70	(3.40)	4.94	(0.90)

Table 6.6. Average items scores on the MEPS in the present study groups and figures reported by Marx et al. (1992).

It would be invalid to directly compare findings from different studies, but the largest difference between the studies that can be seen in the table is between the numbers of relevant means produced by the non-clinical control groups. Marx et al. used a sample drawn from a subject panel affiliated with the Experimental Psychology Department of the University of Oxford, and no details of educational attainment or IQ were reported. It is possible that the community control group used in the present study may have been demographically and cognitively more similar to the clinical groups than was the case for the participants recruited by Marx and colleagues.

Donaldson & Lam (2004) reported a similar approach to that used in the present study for non-clinical recruitment: their control group was recruited through leaflets in the same job centre, and the adjacent post office, in Camberwell, South London. Donaldson and Lam, like the present study, did not find a baseline deficit in problem solving performance by their depressed group compared to controls: the mean total effectiveness score for 2 summed MEPS situations was reported as 7.6 in the depressed group and 7.2 in the control group. A key finding of Donaldson and Lam's study was that a problem solving deficit emerged in the depressed group when they were induced to ruminate. It is possible that among community groups of the sort recruited by Donaldson and Lam, and those taking part in the present study, non-clinical individuals' MEPS responses are not sufficiently more effective than those of depressed people for a difference to be detectable under normal circumstances.

6.2.2.2 Poor AMT and MEPS performance of participants with delusions relative to those without delusions

The pattern of results obtained in the present study using the AMT was similar to that reported by Kaney et al. (1999), who also found OGM in people with persecutory delusions, and not in depressed people without psychosis, compared to non-clinical controls. The present study aimed to systematically examine levels of depression in the groups in relation to memory performance, which Kaney and colleagues did not report. No relationship was found here between depression and OGM. Instead, both subgroups with delusions, regardless of depression level, produced fewer specific memories than the groups without delusions. Problem solving performance followed the same unexpected pattern as AMT scores: no evidence was found for any association with depression, and instead the groups with delusions consistently scored less highly than those without delusions.

AMT and MEPS performance scores were significantly correlated with each other, and with verbal fluency and WTAR scores, which might suggest a common underlying

cognitive component, such as general executive capacity or verbal cognitive processing speed. Meta-analyses of studies in schizophrenia have reported that executive functioning deficits in groups with psychosis tend to be proportional to general IQ and overall performance on a range of cognitive tasks (Heinrichs & Zakzanis, 1998; Henry & Crawford, 2005; Laws, 1999). The participants with delusions in the present study had significantly lower premorbid IQ estimates than the participants without delusions, in line with their lower levels of educational attainment. However, statistically controlling for WTAR scores or verbal fluency in the analysis did not account for the AMT performance difference or the MEPS performance difference between participants with delusions and those without.

Williams and Broadbent's original (1986) AMT study had participants who had all recently attempted suicide. A number of other studies have found specific associations of suicidality, rather than low mood, with AMT and problem solving performance (Schotte & Clum, 1987; Pollock & Williams, 2001; Pollock & Williams, 2004). One study of people with psychosis has linked AMT performance with suicidality (Taylor, Gooding, Wood, & Tarrier, 2010), but with findings in the opposite direction to those of Williams & Broadbent (1986). Taylor et al. found that among a group of people with psychosis, those who had attempted suicide produced *more* specific memories than those who had not attempted suicide. An interpretation was made that within a group with such high levels of difficult experiences and past trauma as tend to be found in people with psychosis, the overgeneral autobiographical memory bias might serve a protective function in decreasing conscious contact with distressing representations of one's past. It is possible that if suicidality and levels of past trauma had been systematically investigated in the present study alongside depression and paranoia, associations with AMT and problem solving performance might have been found.

6.2.3 Jumping to conclusions

The hypothesis was confirmed that people with persecutory delusions were significantly more likely to jump to conclusions on the Beads Task than people without delusions. This finding is in line with those of previous studies of people with persecutory delusions (Corcoran et al., 2008; Startup et al., 2008), and may not apply in the same way to other delusion subtypes (Menon, in press). The present study additionally found that those with delusions who were concurrently depressed were less likely to JTC than those with delusions who were not depressed. This effect of depression on JTC has not been reported before, and could be a characteristic of the present participant sample without necessarily generalising to the wider population.

Nevertheless, the suggestion of an association between problems of affect and data gathering bias within a group of people with delusions is interesting, given that the contributions of these two factors to delusions have been conceptualised as relatively independent (Bentall et al., 2009; Garety et al., 2005).

A further exploratory finding (thus one that may not generalise) was that JTC among the group with delusions was associated with poor problem solving performance on the MEPS. Such a finding could be taken to indicate that hasty data gathering might be causally related to the production of poorly rated problem solving attempts. However, it is also possible that the association was simply a result of both JTC and MEPS performance being related to premorbid IQ. When WTAR scores were controlled for in the analysis, the association between JTC and MEPS performance was no longer significant.

6.2.4 Summary of key cross-sectional findings

Among a group of participants with psychosis who were selected for the presence of persecutory delusions (regardless of affective disturbance), it was found that exactly half met research diagnostic criteria for current major depression. This subgroup reported more negative schematic beliefs about themselves than the subgroup with delusions without depression, as well as higher levels of experiential avoidance and more of a tendency to ruminate in response to distress. This pattern of depression-related cognitive features was also found in a group of people with non-psychotic major depression, relative to a non-clinical control group. Predicted differences between the two subgroups with delusions in memory specificity and problem solving performance were not found, but this might not be described as an absence of depression-related features, because the non-psychotic depressed group also showed no significant deficits in performing these tasks relative to controls. Rather, it appeared that performance on tasks of memory and problem solving was poorer in the participants with delusions than in those without delusions, regardless of depression levels. Low problem solving scores were related to the presence of a JTC reasoning bias and to low premorbid IQ estimates, both of which characterised the participants with delusions compared to the non-psychotic groups. An unexpected finding not previously seen in the literature was that among participants with delusions, co-occurring depression was associated with lower rates of JTC.

Chapter 7. Study Two: longitudinal

In this chapter, the longitudinal results are reported of an investigation that aimed to examine change in the severity of persecutory delusions over six months in the same sample of patients as described in the last two chapters. The primary hypothesis of Study Two was that initial levels of depression and related cognitive factors would predict the persistence of persecutory delusions prospectively over a six month period. It was secondly hypothesised that scores on depression-related cognitive factors would predict the persistence of depression after six months, in line with previous findings in the depression literature.

The severity of persecutory delusions and depression was reassessed dimensionally, and correlation and regression analyses were used to examine hypotheses regarding the prediction of change. The first step was to examine whether baseline depression would predict follow-up delusion severity beyond the explanatory power of baseline delusion severity. Subsequently, specific cognitive factors, which were hypothesised to play a causal role in symptom persistence, were examined in place of depression as predictors. These factors, such as schematic beliefs and problem solving effectiveness, correspond to more specific constructs than the broad constellation of experiences assessed by the BDI, and their statistical associations can be interpreted more meaningfully. Having examined these as predictors of change in delusion severity to test the first hypothesis, the second hypothesis was addressed by evaluating the same factors as predictors of change in depression levels.

7.1 Results

7.1.1 Participant characteristics

Of the 60 Study One participants with persecutory delusions, 54 (90%) were successfully contacted for a follow-up assessment, approximately six months after the first. Some participants were not immediately available for the second appointment, and the time elapsed between the two assessments ranged between 25 weeks and 52 weeks ($M = 33.00$ weeks, $SD = 6.19$ weeks). 44 participants (82%) completed the second assessment within 9 months of the first.

Of the participants successfully followed up, 25 had been in the PD subgroup for Study One, not having met criteria for major depression diagnosis, and 29 had been in the PD+D subgroup with concurrent depression. There were 32 male and 22 female participants. The average age was 41.2 years ($SD = 10.1$ years). The participants

who completed the second assessment did not differ significantly from those who did not on baseline depression (BDI: $t(58) = .92$, $p = .36$) or paranoia (GPTS: $t(58) = -.55$, $p = .59$) scores.

At baseline, 48 (92%) of the 52 participants with available medication data were taking antipsychotic medication. 12 (23%) were on a low dose (up to 200mg chlorpromazine equivalent), 17 (33%) were on a medium dose (201mg to 400mg chlorpromazine equivalent), and 19 (37%) were on a high dose (above 400mg chlorpromazine equivalent). The mean dose of antipsychotic medication taken was 420.38mg chlorpromazine equivalent ($SD = 318.21$ mg). Antidepressants were additionally prescribed to 18 (35%) of the participants, and 19 (37%) participants attended individual or group psychological therapy over the interval between the two assessment points.

7.1.2 Changes in the severity of persecutory delusions

GPTS total scores were the main outcome variable used to measure persecutory delusion severity at each assessment point. Scores relate to overall current severity of paranoia. Changes in PSYRATS scores were also of interest, as a multidimensional measure of changes in the severity of the persecutory delusion that was considered most prominent at baseline.

7.1.2.1 Overall severity changes of persecutory ideation: GPTS scores

At baseline, the mean GPTS total score was 101.69 ($SD = 32.61$), and at follow-up it was 91.13 ($SD = 33.28$). The distributions of GPTS total scores were approximately normal at each assessment point (at baseline, Shapiro-Wilk $w = .98$, $p = .45$; at follow-up, $w = .97$, $p = .22$). Change scores were calculated, and these were converted to percentages of baseline. Figure 7.1 summarises the numbers of participants with GPTS percentage change scores of different magnitudes.

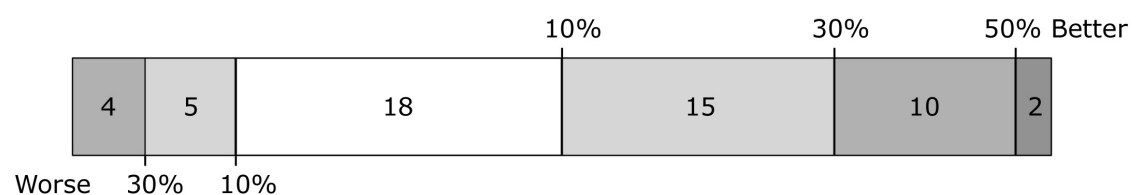


Figure 7.1. Numbers of participants across the range of GPTS percentage change scores.

Of 54 people assessed at both times, 27 (50%) reported paranoia improved by more than 10% at the second assessment point compared to baseline (as indicated by lower GPTS scores). 9 people (17%) reported paranoia increased by more than 10% at the second assessment point compared to the first. Follow-up paranoia scores within 10% of baseline scores were reported by 18 participants (33%). The follow-up scores of 14 participants (26%) differed by more than one standard deviation from baseline: 11 improved, and 3 worsened.

7.1.2.2 Multidimensional severity changes of main persecutory delusions: PSYRATS scores

At baseline, the mean interviewer-rated PSYRATS total score was 15.21 ($SD = 3.76$), and at follow-up it was 13.30 ($SD = 5.65$). The distribution of PSYRATS total scores was approximately normal at baseline (Shapiro-Wilk $w = .98$, $p = .57$), but not at follow-up ($w = .94$, $p = .02$). The follow-up score distribution demonstrated significant negative skew (skewness = -0.65, $SE = 0.33$, $z = -1.97$). Change scores were calculated for each item, and for totals. Figure 7.2 summarises the changes in PSYRATS item scores relating to each participant's main persecutory belief.

Changes were observed across all PSYRATS items. Of the 53 participants with complete ratings at both assessment points, 31 (58%) had improved by at least one PSYRATS point in total over the six month interval. An improvement of at least two PSYRATS points (this was the improvement criterion used by Startup et al., 2007) was recorded for 18 participants (34%). No change in PSYRATS total scores was observed for 11 (21%) participants, and 11 (21%) participants scored at least one point higher on total severity at the follow-up assessment than at baseline.

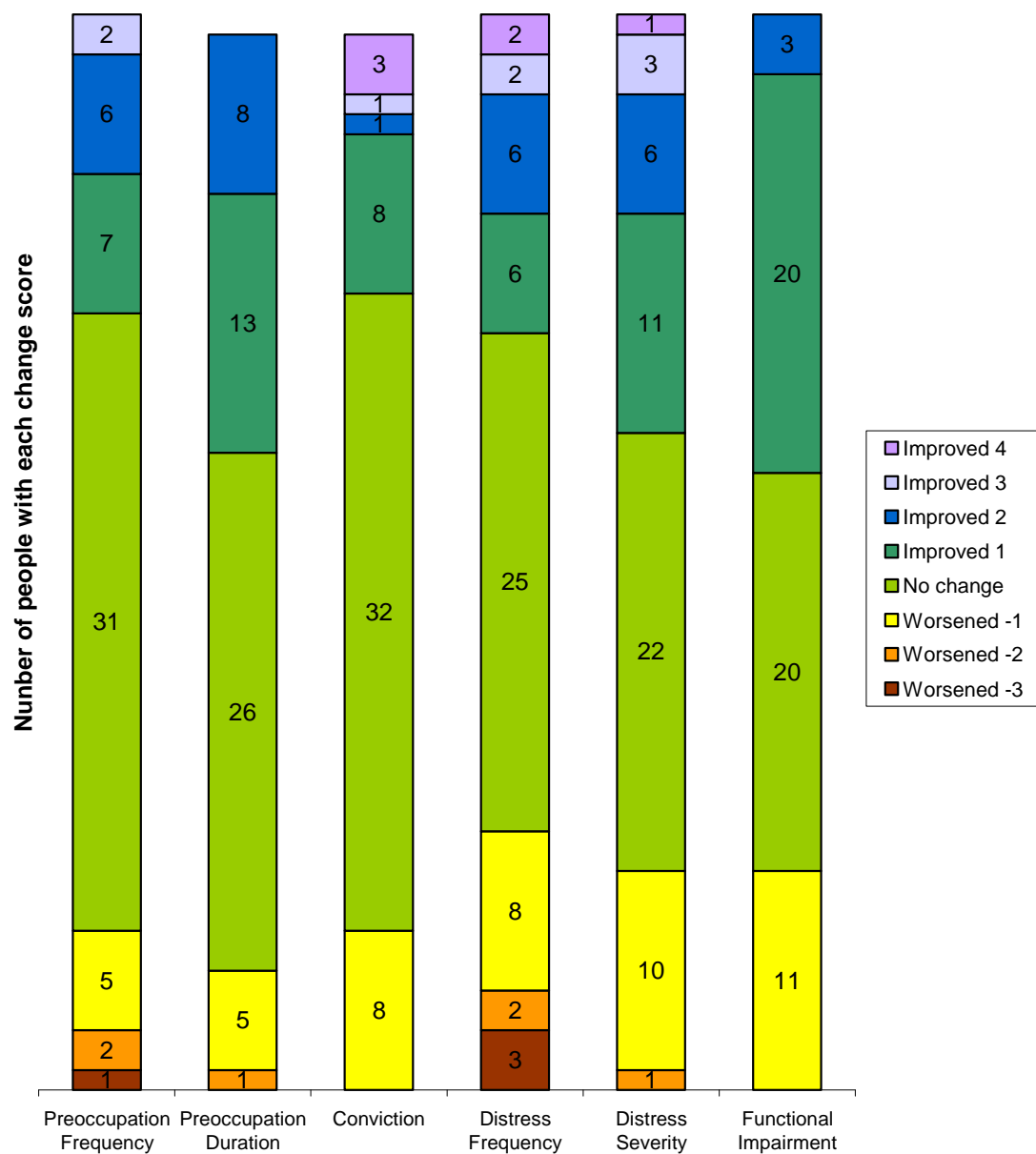


Figure 7.2. Frequency of changes in delusion severity as rated using the PSYRATS.

7.1.2.3 Relationship between GPTS changes and PSYRATS changes

A Shapiro-Wilk test indicated that the distribution of PSYRATS change scores deviated significantly from normality, $w = .87$, $p < .01$. This was associated with significant positive skew (skewness = 1.28, SE = 0.32, $z = 3.91$) and leptokurtosis (kurtosis = 2.39, SE = 0.64, $z = 3.71$). The distribution of GPTS change scores did not deviate significantly from normality according to the Shapiro-Wilk test, $w = .98$, $p = .36$. Negative skew was non-significant (skewness = -0.34, SE = 0.33, $z = -1.03$), and

leptokurtosis was also non-significant (kurtosis = 0.40, SE = 0.64, $z = 0.62$). GPTS change scores were judged to be suitable for parametric correlation analysis, and PSYRATS change scores were not. A non-parametric Spearman's correlation indicated a significant positive association between PSYRATS change scores and GPTS change scores, $r_s = .32$, $p = .02$.

7.1.3 Predicting changes in levels of persecutory ideation

Figure 7.3 shows a scatter plot of the participants' GPTS scores at baseline and at six month follow-up, with a reference line indicating equal scores at the two assessments. Points below the line correspond to participants who showed improvement over the interval, and points above the line correspond to participants with increases in paranoia severity over time. The correlation between GPTS baseline and follow-up scores was significant and high, $r = .65$, $p < .01$, corresponding to $r^2 = 42\%$ shared variance between the two measurements.

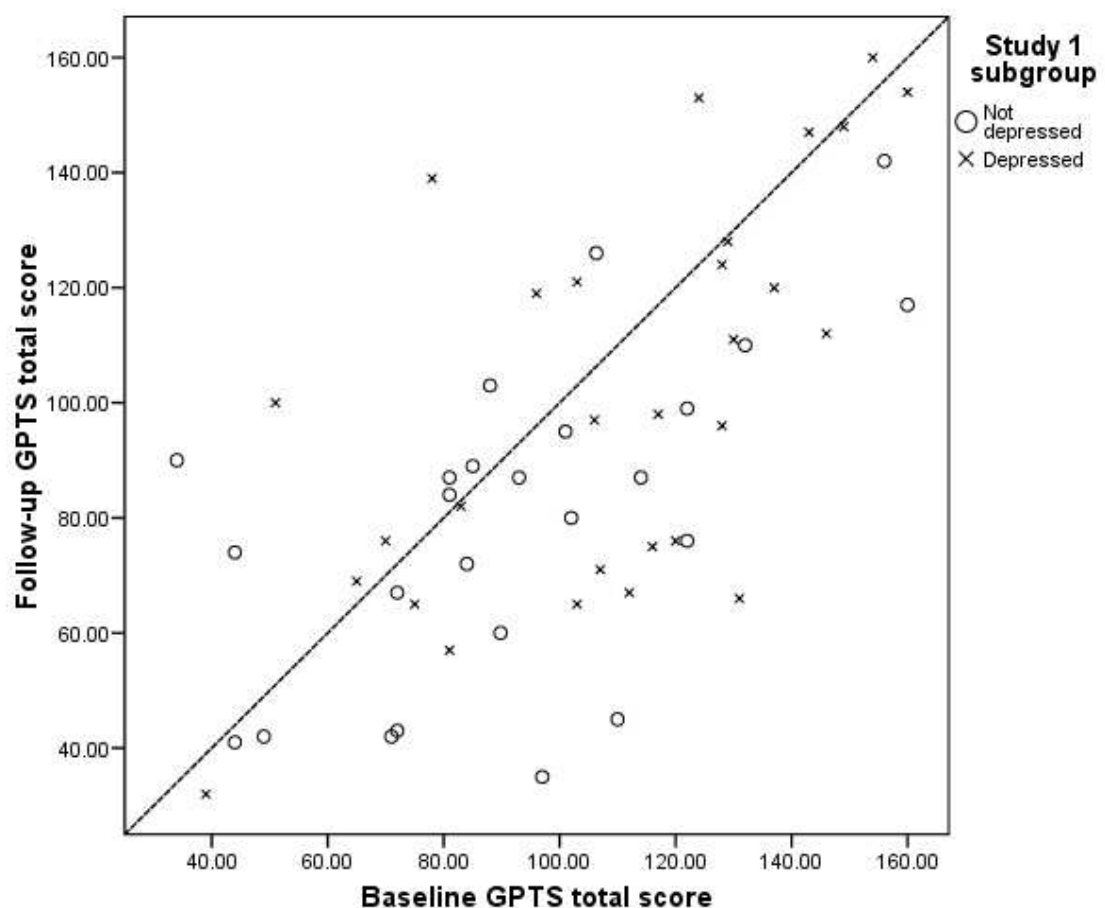


Figure 7.3. Scatter plot of participants' Green et al. Paranoid Thoughts Scale (GPTS) scores at baseline and follow-up assessment. Reference line indicates equal scores.

7.1.3.1 Age, sex and treatment effects

Partial correlations were computed to examine any significant effects of age, sex or treatment on follow-up GPTS scores, while controlling for baseline scores. Categorical variables can be used in correlation analysis when they only have two levels: such variables here were sex and whether or not the participant had undergone psychological therapy in the period between assessments. No significant partial correlations were found of follow-up GPTS scores with age ($r = .08$, $p = .58$), sex ($r = -.11$, $p = .46$), antipsychotic medication chlorpromazine equivalent dose ($r = .27$, $p = .06$), antidepressant medication use ($r = .11$, $p = .45$), or psychological therapy ($r = -.14$, $p = .34$). It was therefore deemed inappropriate to control for such variables in the further analyses, in view of the loss of power associated with such statistical procedures.

7.1.3.2 Partial correlations with depression and cognitive factors

The prime longitudinal hypothesis of the present study was that levels of depression and associated cognitive factor scores would significantly predict additional variance in GPTS follow-up scores beyond that which could be predicted from baseline scores. In order to test this hypothesis, partial correlations were computed between follow-up GPTS scores and each of the predictors of interest, controlling for baseline GPTS scores. This would reveal any significant associations with prognosis. Alongside depression and cognitive predictors, BAI anxiety scores were also included in the partial correlation analysis, in recognition of previous research interest in the role of anxiety in persecutory delusions, as well as its documented association with depression. Table 7.1 gives the resulting partial correlation coefficients, with associated significance values. Significant partial correlations were found for depression, anxiety, negative schematic beliefs about the self, MEPS total means scores, MEPS effectiveness scores and worry. All were of medium effect size.

Variable	Partial correlation with follow-up GPTS scores
Depression (BDI)	$r = .42, p < .01^{**}$
Anxiety (BAI)	$r = .32, p = .02^*$
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>	
Self Negative	$r = .37, p < .01^{**}$
Self Positive	$r = -.17, p = .22$
Other Negative	$r = -.06, p = .66$
Other Positive	$r = -.06, p = .68$
Avoidance (AAQ)	$r = .06, p = .67$
Rumination (RRS)	$r = .21, p = .14$
Memory specificity (AMT)	$r = -.17, p = .23$
<i>Means-Ends Problem Solving (MEPS)</i>	
Total means	$r = -.33, p = .02^*$
Effectiveness	$r = -.31, p = .03^*$
Worry (PSWQ)	$r = .34, p = .01^*$

Table 7.1. Partial correlations of baseline predictors with follow-up GPTS scores, controlling for baseline GPTS scores.

*** indicates significance at $p < .01$ level; * indicates significance at $p < .05$ level.*

7.1.3.3 Multicollinearity among predictors

In order to obtain a stable and meaningful multiple regression model, it is desirable that predictor variables are not highly intercorrelated. Table 7.2 summarises the zero-order correlation coefficients, with associated significance values, between paranoia measurements, baseline depression, and the significant cognitive predictors of paranoia that were identified using partial correlation.

	Follow-up GPTS	Baseline GPTS	BDI	BCSS SN	MEPS M	MEPS E
	.65**					
Baseline GPTS	< .01					
	.60**	.49**				
Depression (BDI)	< .01	< .01				
BCSS Self Negative	.58**	.49**	.83**			
	< .01	< .01	< .01			
<i>Means-Ends Problem Solving (MEPS)</i>						
	-.25	.04	-.02	-.02		
Total means	.07	.75	.90	.87		
	-.26	-.01	-.11	-.12	.93**	
Effectiveness	.06	.96	.41	.35	< .01	
	.45**	.36**	.60**	.51**	.05	.07
Worry (PSWQ)	< .01	< .01	< .01	< .01	.73	.61

Table 7.2. Zero-order Pearson's correlation coefficients between paranoia measurements and predictors. Significance values are given below coefficients.

*** indicates significance at $p < .01$ level.*

Very high correlations ($r > .8$) were indicated between BDI and BCSS-SN scores, and between the two MEPS scoring measures, indicating such a high degree of shared variance that both variables in such a correlated pair should not be used together as predictors in a regression model (Field, 2009, p. 224). Worry scores were also highly correlated ($r > .5$), albeit to a lesser degree, with BDI and BCSS-SN scores.

7.1.3.4 Depression predicting changes in persecutory delusion severity

In order to evaluate any significant increases in R^2 associated with the addition of depression to a model predicting change in GPTS scores over time, a hierarchical linear regression model was run with follow-up GPTS scores as the dependent variable, baseline GPTS scores as predictor at the first step and BDI scores as additional predictor at the second step. Table 7.3 gives the model coefficients.

	<i>B</i>	<i>SE B</i>	β	<i>p</i>
Step 1				
Constant	23.94	11.51		
Baseline GPTS	0.66	0.11	.65	< .01
Step 2				
Constant	22.66	10.57		
Baseline GPTS	0.48	0.11	.47	< .01
Baseline BDI	0.81	0.25	.37	< .01
$R^2 = .42$ for Step 1, $\Delta R^2 = .10$ for Step 2 ($p < .01$).				

Table 7.3. Hierarchical multiple regression predicting follow-up GPTS scores using baseline BDI scores.

At the first step, the model using just baseline GPTS scores explained, based on the adjusted R^2 , 41% of the follow-up GPTS variance, $F(1, 52) = 37.53$, $p < .01$. Where the model R^2 statistic indicates how much dependent variable variation in the present sample can be explained by the model, the *adjusted* R^2 is an estimate of the broader population variance that might be explained by the model, when its complexity and multicollinearity are taken into account. Models with more predictor variables and high multicollinearity tend to have greater downward adjustments in R^2 , and might be expected to show poorer cross-validity in the broader population than more parsimonious models and those with more orthogonal predictors.

The addition of BDI scores to the model led to a significant increase in R^2 at the second step, explaining a further 10% of the variance in follow-up GPTS scores over that explained by baseline GPTS scores, $F(1, 51) = 10.72$, $p < .01$. The final model had significant fit to the data, $F(2, 51) = 27.63$, $p < .01$, with adjusted $R^2 = .50$. Each of

the two factors in the final model significantly predicted the outcome with the other factor controlled.

7.1.3.5 Cognitive factors predicting changes in persecutory delusion severity

Specific cognitive factors were used in place of depression in a hierarchical regression model as described above, so that hypothesised associations of particular cognitive mechanisms with delusion persistence could be evaluated. The factors identified as significant using partial correlations in section 7.1.3.2 were entered as predictors. Due to the very high correlation between the two MEPS measures, which are likely to measure to a high degree the same underlying construct, just the total relevant means scores were used in this analysis. The first step was identical to that described in section 7.1.3.4. At the second step, BCSS self negative belief scores, MEPS total means scores and PSWQ worry scores were added as predictors. Table 7.4 gives the model coefficients. The coefficients at the first step differ somewhat from those in Table 7.3, because one participant with missing PSWQ data was excluded from the present analysis.

	<i>B</i>	<i>SE B</i>	<i>β</i>	<i>p</i>
Step 1				
Constant	19.94	10.99		
Baseline GPTS	0.69	0.10	.69	< .01
Step 2				
Constant	20.05	16.73		
Baseline GPTS	0.53	0.11	.53	< .01
BCSS self negative	0.85	0.58	.18	.15
MEPS total means	-1.92	0.76	-.23	.02
PSWQ worry	0.50	0.27	.20	.07
<i>R</i> ² = .47 for Step 1, ΔR^2 = .14 for Step 2 (<i>p</i> < .01).				

Table 7.4. Hierarchical multiple regression predicting follow-up GPTS scores using baseline cognitive factor scores.

At the first step, the model using just baseline GPTS scores explained, based on the adjusted R^2 , 46% of the follow-up GPTS variance, $F(1, 51) = 45.00$, $p < .01$. The addition of the three cognitive factors to the model led to a significant increase in R^2 at the second step, explaining a further 14% of variance in the participants' follow-up GPTS scores over that explained by baseline GPTS scores, $F(3, 48) = 5.45$, $p < .01$. The final model had significant fit to the data, $F(4, 48) = 18.29$, $p < .01$, with adjusted $R^2 = .57$. Baseline GPTS scores and MEPS total means scores each significantly

predicted follow-up GPTS scores with the other factors held constant, but BCSS self negative beliefs and PSWQ worry did not.

The primary hypothesis was supported by the finding that levels of self-reported depression and several specific cognitive factors linked with affective disturbance significantly predicted follow-up levels of persecutory ideation beyond the predictive power of baseline severity. Depression, negative schematic beliefs about the self, poor problem solving performance and worry all showed significant predictive associations of medium effect size. Hierarchical multiple regression analysis indicated that baseline depression levels enabled 10% of the variation in follow-up paranoia to be explained on top of what could be predicted by baseline paranoia scores. An alternative model indicated that the three significantly predictive cognitive factors together enabled 14% of follow-up paranoia variance to be predicted beyond that accounted for by baseline paranoia levels.

7.1.4 Changes in levels of depression

At baseline, the mean BDI score among 54 participants was 24.96 ($SD = 15.6$), and at follow-up it was 23.85 ($SD = 16.47$). The distribution of BDI scores at baseline demonstrated significant positive skew (skewness = 0.78, $SE = 0.33$, $z = 2.39$); the distribution at follow up showed non-significant positive skew (skewness = 0.58, $SE = 0.33$, $z = 1.79$). Change scores were calculated and converted to percentages of baseline. Figure 7.4 summarises the numbers of participants with BDI percentage change scores of different magnitudes.

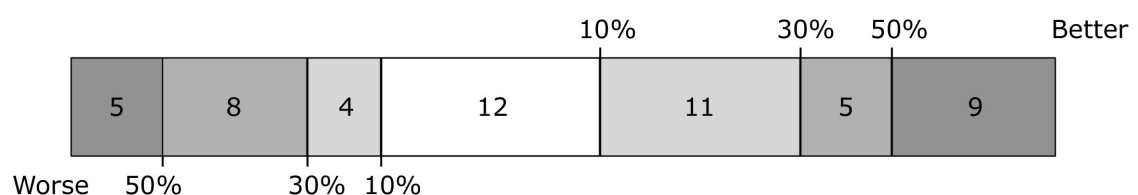


Figure 7.4. Numbers of participants across the range of BDI percentage change scores.

Of the 54 participants successfully followed up, 25 (46%) had BDI depression scores reduced by more than 10% at follow-up compared to baseline, 17 (31%) had depression scores increased by more than 10%, and 12 participants (23%) scored within 10% in either direction of their baseline score at follow-up. The follow-up scores of 4 participants (7%) differed by more than one standard deviation from baseline: 2 improved, and 2 worsened.

7.1.5 Predicting changes in levels of depression

Figure 7.5 shows a scatter plot of baseline and follow-up BDI scores, with a reference line indicating equal scores at the two assessment points. BDI scores at the two assessment points were highly correlated, $r = .87$, $p < .01$, corresponding to 75% shared variance.

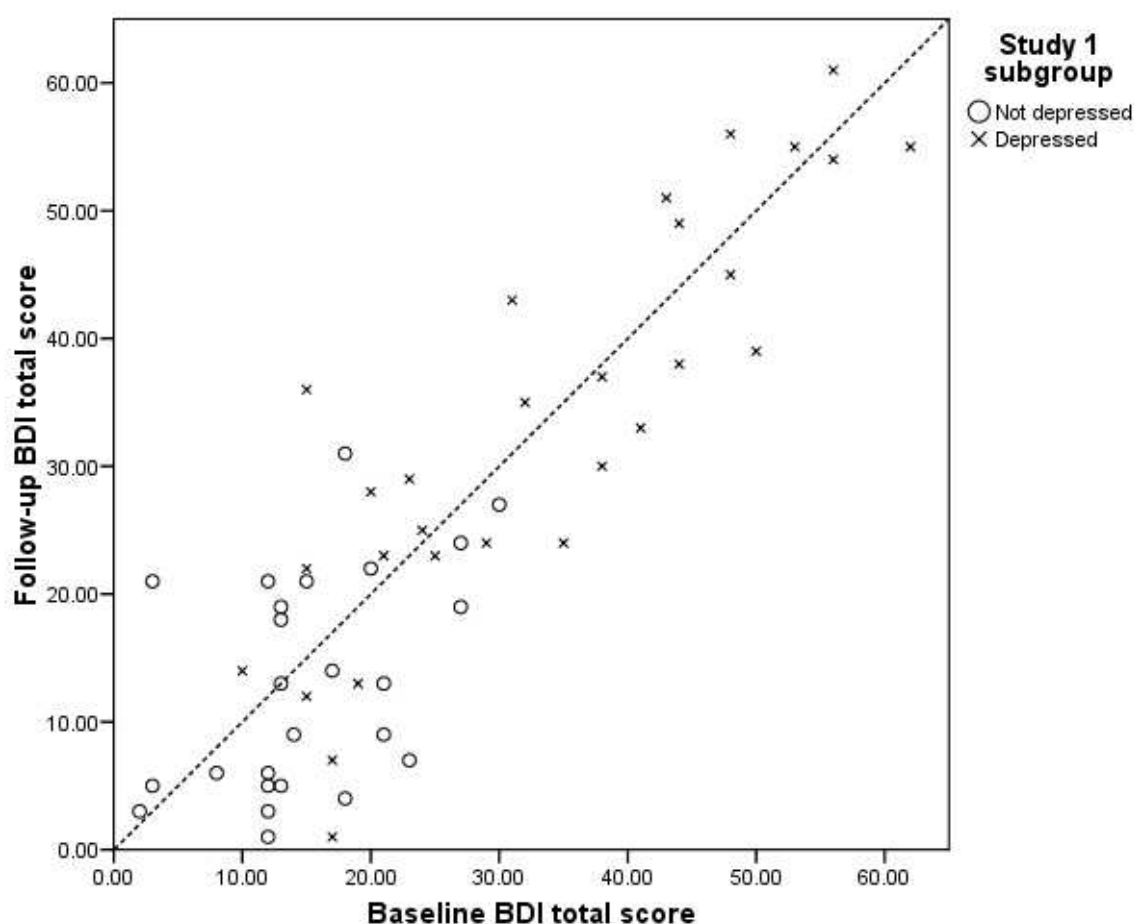


Figure 7.5. Scatter plot of participants' Beck Depression Inventory (BDI) scores at baseline and follow-up assessment. Reference line indicates equal scores.

7.1.5.1 Age, sex and treatment effects

Partial correlations were computed to examine any significant effects of age, sex or treatment on follow-up BDI scores, while controlling for baseline scores. No significant partial correlations were found of follow-up BDI scores with age ($r = .04$, $p = .79$), sex ($r = .04$, $p = .79$), antipsychotic medication chlorpromazine equivalent dose ($r = .06$, $p = .70$), antidepressants ($r = .01$, $p = .94$), or psychological therapy ($r = -.01$, $p = .95$). It was therefore deemed inappropriate to control for such variables in further analyses.

7.1.5.2 Cognitive factors predicting changes in depression levels

The second longitudinal hypothesis was that baseline scores on depression-related cognitive factors would predict the persistence of depression over the follow-up period. To test this hypothesis, partial correlations were computed between follow-up BDI scores and cognitive predictor variables, with baseline BDI scores entered as a covariate. This would identify any factors that might account for significant variation in follow-up depression scores, beyond that accounted for by baseline scores.

Partial correlations indicated significant associations of follow-up BDI scores with BCSS self negative belief scores and with both MEPS problem solving scores. Table 7.5 gives the partial correlation coefficients for all of the variables examined.

Variable	Partial correlation with follow-up BDI scores
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>	
Self Negative	$r = .34, p = .01^*$
Self Positive	$r = .20, p = .15$
Other Negative	$r = .12, p = .39$
Other Positive	$r = .16, p = .27$
Avoidance (AAQ)	$r = .09, p = .53$
Rumination (RRS)	$r = .02, p = .90$
Memory specificity (AMT)	$r = -.20, p = .15$
<i>Means-Ends Problem Solving (MEPS)</i>	
Total means	$r = -.33, p = .02^*$
Effectiveness	$r = -.32, p = .02^*$
Worry (PSWQ)	$r = .08, p = .58$

Table 7.5. Partial correlations of baseline predictors with follow-up BDI scores, controlling for baseline BDI scores.

* indicates significance at $p < .05$ level.

In order to evaluate any significant increases in R^2 associated with the addition of cognitive factors to a model predicting change in BDI scores over time, a hierarchical linear regression model was run with follow-up BDI scores as the dependent variable, baseline BDI scores as predictor at the first step and cognitive predictors added at the second step. BCSS self negative belief scores and MEPS total means scores were used as cognitive predictors. Table 7.6 gives the model coefficients.

	<i>B</i>	<i>SE B</i>	β	<i>p</i>
Step 1				
Constant	0.17	2.19		
Baseline BDI	0.95	0.08	.87	< .01
Step 2				
Constant	7.17	3.16		
Baseline BDI	0.65	0.13	.59	< .01
BCSS self negative	0.76	0.29	.32	.01
MEPS total means	-0.67	0.26	-.16	.01
$R^2 = .75$ for Step 1, $\Delta R^2 = .05$ for Step 2 ($p < .01$).				

Table 7.6. Hierarchical multiple regression model predicting follow-up BDI scores.

At the first step, the model using just baseline BDI scores explained, based on the adjusted R^2 , 75% of the follow-up BDI variance, $F(1, 52) = 158.06$, $p < .01$. The addition of the two cognitive factors to the model led to a significant increase in R^2 at the second step, explaining a further 5% of the variance in follow-up BDI scores over that explained by baseline BDI scores, $F(2, 50) = 6.94$, $p < .01$. The final model had significant fit to the data, $F(3, 50) = 69.36$, $p < .01$, with adjusted $R^2 = .80$. Baseline BDI scores, BCSS self negative belief scores and MEPS total means scores each significantly predicted follow-up BDI scores with the other factors held constant.

The second hypothesis was supported by the finding that scores on cognitive factors predicted follow-up depression scores over and above the substantial variance that could be predicted by baseline depression scores. Higher follow-up depression scores were significantly predicted by greater negative schematic beliefs about the self and poorer problem solving performance at baseline.

7.1.6 Changes in cognitive factor scores

Table 7.7 shows descriptive statistics for the main cognitive factor scores at each of the two assessment points.

N = 54*	Baseline M	(SD)	Follow-up M	(SD)
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>				
Self Negative	8.78	(6.88)	8.39	(6.67)
Self Positive	8.87	(6.28)	8.96	(6.76)
Other Negative	13.13	(6.98)	13.44	(7.04)
Other Positive	8.93	(6.58)	8.93	(6.60)
Avoidance (AAQ)	40.98	(8.06)	40.73	(8.20)
Rumination (RRS)	54.39	(15.12)	54.00	(13.65)
<i>Autobiographical Memory Task (AMT) alternate forms</i>				
Total specificity	5.83	(2.90)	5.85	(3.13)
<i>Means-Ends Problem Solving (MEPS) alternate forms</i>				
Total means	9.22	(3.91)	15.57	(7.70)
Effectiveness	10.04	(2.81)	11.54	(3.27)
Worry (PSWQ)	53.11	(13.03)	54.94	(13.87)

Table 7.7. Means and standard deviations (in parentheses) of cognitive factor scores at the two assessment points.

** Baseline AAQ N=52, Baseline PSWQ N=53, Follow-up AAQ N=51, Follow-up PSWQ N=52.*

The similarity of each pair of figures indicated considerable stability over time. The only measure showing a mean change bigger than one SD was the MEPS total means score, which might have been affected by the different problem situations used in the two alternate forms (these had not been matched for difficulty).

7.1.7 Associations between symptom changes and cognitive changes

Change scores were calculated for the main symptom measures (GPTS; BDI) and cognitive variables by subtracting each participant's follow-up score from their baseline score. Correlation coefficients were calculated between change scores on symptom measures and cognitive factors. Table 7.8 shows these correlation coefficients and associated significance values.

N = 54*	Paranoia (GPTS)	(p)	Depression (BDI)	(p)
Depression (BDI)	.39	< .01		
<i>Schematic beliefs: Brief Core Schema Scales (BCSS)</i>				
Self Negative	.29	.03	.14	.31
Self Positive	-.14	.31	-.43	< .01
Other Negative	.41	< .01	.03	.84
Other Positive	.02	.89	-.10	.48
Avoidance (AAQ)	.13	.37	.27	.06
Rumination (RRS)	.12	.38	.38	< .01
<i>Autobiographical Memory Task (AMT)</i>				
Total specificity	-.06	.65	-.05	.70
<i>Means-Ends Problem Solving (MEPS)</i>				
Total means	-.07	.63	-.01	.94
Effectiveness	.09	.51	.17	.22
Worry (PSWQ)	-.15	.31	.28	.05

Table 7.8. Pearson's *r* correlation coefficients and significance values (in parentheses) between change scores of symptoms and cognitive factors.

*AAQ N=50, PSWQ N=51.

Significant positive associations were indicated between paranoia changes and changes in negative beliefs about the self and negative beliefs about others, such that increases in paranoia were associated with increases in negative beliefs. Significant positive associations were indicated between depression changes and changes in rumination and worry, such that increases in depression were associated with increases in the cognitive factor scores. Additionally, a significant negative association was found between changes in depression and changes in positive beliefs about the self, such that increases in depression were associated with decreases in positive beliefs about the self, and vice versa. Changes in paranoia scores and depression scores were also significantly correlated with each other, such that symptom improvements in one domain were associated with improvements in the other. All of these associations were of approximately medium effect size.

7.2 Discussion

The two longitudinal study hypotheses concerned prediction of symptom changes over time. In order to test these hypotheses, it was crucial that symptom changes were detected over the follow up period.

7.2.1 Describing symptom changes

Most participants' GPTS paranoia follow-up scores differed from their baseline scores by more than 10%, and a quarter of the scores changed by more than one standard deviation. Most participants' BDI depression scores also differed from baseline by more than 10%, but only 4 of 54 changed by more than a standard deviation. It is possible that the depression SD was relatively large compared to the paranoia SD, because the present participants were selected for clinically high paranoia, while depression levels were not controlled.

PSYRATS scores indicated changes on every dimension of delusion severity, including preoccupation, conviction, distress and functional impairment. A third of participants (34%) showed improvements of two points or more in total over the follow-up period. Startup et al. (2007) reported that 24% of their participant sample showed this magnitude of improvement over three months.

GPTS and PSYRATS change scores were significantly correlated, indicating that changes in the severity of specific, prominent, operationally defined (Freeman & Garety, 2000) persecutory delusions were associated with changes in the same direction in the overall severity of paranoid ideation. This gives convergent support for the validity of measuring persecutory delusion change using the GPTS.

7.2.2 Predicting changes in persecutory delusion severity

The main hypothesis of the study was that depression and associated cognitive factors would predict the persistence of persecutory delusions over time. This hypothesis was supported by findings that follow-up severity of persecutory ideation was significantly predicted, beyond what could be explained by baseline severity, by baseline levels of depression and cognitive factor scores. The following cognitive predictors were chosen for this investigation because of their prominence in the depression literature: schematic beliefs, experiential avoidance, rumination, autobiographical memory bias and social problem solving performance. Worry was also included, because previous research indicated an association between this process and the persistence of delusions (Startup et al., 2007). Results indicated that negative schematic beliefs about the self, social problem solving deficits and worry each significantly predicted higher follow-up persecutory delusion severity beyond what could be accounted for by baseline severity scores, all with medium effect size. No significant predictive effects were found of other subtypes of schematic belief, avoidance, rumination or memory bias.

7.2.2.1 Depression predicts persistence of persecutory delusions

Depression alone was found to significantly predict 10% of variation in follow-up paranoia severity, on top of what could be predicted by baseline severity scores. The association was positive, such that at every given level of baseline paranoia severity, higher baseline depression scores predicted higher follow-up paranoia scores. This is in line with the findings of previous studies reviewed in Chapter 2 (Bergstein et al., 2008; Drake et al., 2004; Fowler et al., 2011; Thewissen et al., 2011), and contributes to mounting evidence that depression in people with persecutory delusions is associated with impaired recovery.

The complexity of depression can impair the interpretation of its role to some extent. The BDI assesses a range of cognitive, affective and somatic symptoms, which are typically summed to a total severity score. Items relating to sleep and appetite have equal weighting to those about low mood and suicidal thoughts. It is of interest whether certain features of depression might play a particularly significant role, and this cannot be deduced from associations of total summed scores. It is common to divide the BDI into two subscales: a cognitive subscale and an affective-somatic subscale (Steer, Ball, Ranieri, & Beck, 1999; Beck et al., 1996). The selection of cognitive process measures included in the present study allowed even more specific associations to be examined.

7.2.2.2 Cognitive factors predict persistence of persecutory delusions

Negative schematic beliefs, problem solving scores and worry were each significantly predictive of the persistence of persecutory delusions over the follow-up period. A combination of these three factors was found to significantly predict 14% of variation in follow-up paranoia severity, beyond what could be predicted by baseline severity scores. At every given level of baseline paranoia severity, more negative beliefs about the self, poorer problem solving and more worry predicted higher follow-up paranoia severity.

The main novel finding of the present study was that low problem solving performance scores predicted the persistence of persecutory delusions over time. This is the first known study of social problem solving in a group of people specifically selected for the presence of delusions. Platt and Spivack's early studies of the MEPS (Platt & Spivack, 1972a; Platt & Spivack, 1972b) used mixed groups of psychiatric inpatients dominated by those with schizophrenia, but clinical symptom profiles were not reported, and psychiatric control groups were not used, so the results said little about the relationship between performance deficits and any particular symptom dimension.

Social problem solving deficits have also been shown in groups of participants with bipolar disorder, depression and anxiety disorders (Bellack, Sayers, Mueser, & Bennett, 1994; Marx et al., 1992). Platt and Spivack interpreted their original findings in terms of a broad problem-solving approach to psychopathology, whereby an impaired ability to generate appropriate solutions to life problems drives the functional impairment of the maladjusted individual. Williams' (2006) model of depressive cognitive processes also proposed that a range of information processing biases exert their effects on symptom persistence via interference with problem solving ability.

The results of the present study suggest that people with persecutory delusions experience less improvement of paranoia over time if they perform less well on problem solving tasks. This novel finding is interpreted with caution. In particular, causality cannot be inferred from an observational study such as this. It is possible that other, unmeasured variables underlie the statistical associations between study measures. Given the present results, it is plausible that problem solving difficulties might contribute to the maintenance of persecutory delusions and depression over time, for example by affecting instrumental behaviours and leading the individual into unhelpful situations. Conversely, it might be that those individuals who are not going to improve have lower problem solving scores for a different reason, for example that they have more severe pathology to begin with. However, in the present sample of participants, problem solving scores were not significantly correlated with paranoia or depression levels, and changes in problem solving scores did not correlate with symptom change scores. Problem solving scores were thus relatively independent of symptom severity, yet predicted the persistence of both paranoia and depression over time.

Consistent with the proposal that problem solving deficits can contribute to the persistence of persecutory delusions, there is an indication in the literature that problem solving therapy might lead to significant improvements in psychotic symptoms, including delusions (Tarrier et al., 1993).

The finding that negative schematic beliefs about the self predicted the persistence of paranoia replicates the results of Fowler et al. (2011), suggesting that this effect can be detectable even with considerably fewer participants, a shorter follow-up period and a less sophisticated analysis technique. The finding that worry predicted the persistence of delusions replicates the results of Startup et al. (2007), extending the effect over a further three months. Startup et al.'s additional finding that anxiety predicted the persistence of delusions was also replicated here. The focus of the

present study was on processes related to depression, but this is not to suggest that these features are specific to just depression, or that other symptom dimensions are unimportant. The role of depressive processes in delusion persistence is conceptualised within a multifactorial framework, as described in Chapter 1, alongside other factors including anxiety and reasoning bias.

7.2.2.3 Negative findings for experiential avoidance, rumination and memory specificity

No significant association with persecutory delusion persistence was found for experiential avoidance, rumination or memory specificity. None of these factors are known to have been previously investigated as predictors of paranoia persistence over time, with the exception of Udachina et al.'s (2009) experience sampling study, which reported that over short periods of time (across successive beep assessments, where beeps occurred ten times a day for six consecutive days) high experiential avoidance predicted subsequent paranoia increases. Udachina et al.'s study concerned subclinical paranoia in a sample of students. The present study found no evidence to support a predictive effect of experiential avoidance on the persistence of paranoia in people with persecutory delusions. Udachina et al. used a revised version of the Acceptance and Action Questionnaire (AAQ-II; Bond et al., 2011), which had not been published and was not known to the authors of the present study at the time that measures were selected. The AAQ-II was devised as an improvement on the first AAQ, with the aim of addressing some criticisms regarding the understandability of items and the scale's psychometric properties. It is possible that if the AAQ-II had been used in the present study instead of the original AAQ, significant predictive effects might have been observed.

Rumination showed a non-significant association with delusion persistence (partial correlation $r = .21$, $p = .14$), which might have reached significance in a larger sample. The related construct of worry, which also involves repetitive negative thinking, but with somewhat different content, did demonstrate a significant predictive effect. It has been proposed that negative repetitive thought is a transdiagnostic pathological mental process involved in most Axis 1 disorders, but that the measures of rumination and worry used here – the Ruminative Response Scale and the Penn State Worry Questionnaire – are not well suited for measuring repetitive negative thought in people with diagnoses other than depression and generalized anxiety disorder, respectively, because both focus on disorder-specific thought content (Ehring & Watkins, 2008; Ehring et al., 2011). Ehring and colleagues proposed that a content-free measure of perseverative thought would be less vulnerable to bias by particular symptoms (Ehring

et al., 2011). The present study employed the most widely used measures of rumination and worry, in order that the findings would be comparable to those of previous research. The finding that worry significantly predicted the persistence of paranoia, but rumination did not, illustrates the differential performance of perseverative thought measures with different content. It is possible that, for people with persecutory delusions, “worry” forms of repetitive thought, which tend to be anxious and future-orientated, are more relevant than “rumination” forms of repetitive thought, which tend to be past-orientated and sad rather than fearful.

Memory specificity, measured using the Autobiographical Memory Task, showed no significant relationship with the persistence of paranoia in the present study. No other study is known to have examined longitudinal symptom prediction using the AMT in a group of people with delusions. The three known cross-sectional studies that measured AMT performance of groups with psychosis all obtained quite different findings (Kaney, Bowen-Jones, & Bentall, 1999; Iqbal, Birchwood, Hemsley, Jackson, & Morris, 2004; Taylor et al., 2010), as outlined in Chapter 4. No empirical support has been found for an effect of memory specificity on the persistence of psychotic symptoms.

7.2.3 Predicting changes in depression severity

The second hypothesis was that depression persistence would be predicted by cognitive factors selected from previous research on major depressive disorder. The five factors examined as predictors were schematic beliefs, experiential avoidance, rumination, autobiographical memory bias and social problem solving performance. The hypothesis was partially supported by a similar pattern of findings to that reported for paranoia. Negative schematic beliefs about the self and problem solving scores predicted follow-up depression severity, beyond the predictive power of baseline severity scores. Experiential avoidance, rumination and memory specificity were not found to predict depression persistence.

All five factors selected for this analysis have been found to predict the persistence of depression over time, but no previous studies are known to have examined this in a group of people with persecutory delusions. The results of the present study suggest that negative schematic beliefs about the self and problem solving difficulties could perpetuate depression occurring alongside persecutory delusions in a similar way to that proposed for major depressive disorder. This is consistent with Fowler et al.'s (2011) finding that negative schematic beliefs about the self and low mood were reciprocally associated over time in a group of participants who recently experienced

a psychotic relapse. No support was found in the present study for an association of experiential avoidance, rumination or memory bias with depression persistence. Rumination change scores correlated significantly with depression change scores, and the experiential avoidance change scores' correlation with depression change scores approached significance, which suggests that these two factors had some association with symptom levels. It might be that experiential avoidance and rumination in this group act as state-like markers of depression and not as predictors of change. Memory specificity scores did not demonstrate any significant association with depression persistence, and change scores were not significantly correlated with symptom changes. The role that overgeneral memory bias is thought to play in the perpetuation of depression (Williams et al., 2007) may not apply in the same way to depression occurring alongside persecutory delusions.

7.3 Summary

The main hypothesis was supported by findings that the persistence of paranoia over time was significantly predicted by depression and by two of the five hypothesised cognitive predictors: negative schematic beliefs about the self and problem solving difficulties. The significance of depression and of negative schematic beliefs about the self in predicting the persistence of paranoia has been reported in several previous studies, and the present findings accrue to support these effects. Previous findings that worry predicts paranoia persistence were also replicated.

The second hypothesis was also supported by significant findings for two of five hypothesised predictors. The same two cognitive factors - negative schematic beliefs about the self and problem solving difficulties - were found to significantly predict the persistence of both paranoia and depression.

The next chapter will bring together the cross-sectional findings of the previous chapter and the longitudinal results described here, integrate them in the context of previous findings and reflect on theoretical and possibly clinical implications. The limitations of the present research will also be summarised with reference to some possible uncontrolled sources of error, and some directions for future investigation will be indicated.

Chapter 8. Integrated discussion and conclusions

It has been demonstrated that depression is common in people with psychosis, and that the burden of additional morbidity that it carries is significant. Despite widespread acknowledgement of the scale of this problem, there is no agreed management strategy, and clinicians' widely discordant treatment approaches reflect different beliefs about the aetiology of depressive symptoms occurring in this context (Addington et al., 2002; Upthegrove, 2009).

The dominant view that depression is, in one way or another, secondary to psychotic disorder may have obscured investigation of the opposite effect, proposed among others by Garety et al. (2001): that depression and its antecedents can drive the occurrence or persistence of positive psychotic symptoms. The present thesis set out to examine such an effect, focusing on Freeman et al.'s (2002) model of the persistence of persecutory delusions.

The prevalence of clinical depression in participants with persecutory delusions was substantial, and corresponded closely to previous reports concerning symptomatically unspecified groups of people with schizophrenia-spectrum disorders. It was shown that depression in this context shares a number of cognitive features with major depressive disorder, and that high levels of depression predict the persistence of persecutory delusions over time.

Evidence of shared cognitive features not only helps to identify depression in this group as similar to that seen in major depressive disorder, but suggests mechanisms that might connect affective disturbance with psychosis. Freeman et al.'s (2002) account proposed that certain cognitive features of depression can contribute directly to the persistence of persecutory beliefs, while other cognitive factors can predispose individuals to both paranoia and depression. This chapter brings together cross-sectional and longitudinal findings for each of the putative cognitive mechanisms, which are examined against the predictions of theoretical models. Some possible implications for clinical practice and directions for further research are discussed.

8.1 The relationship between depression and psychosis

"Everything is a bit jaded. Everything is procrastinated or postponed. Procrastination, delay, a kind of fretting enters into the picture. Because your better sense of yourself is taken away, by the anguish and the psychosomatic intrusion. In an immediate

personal sense, your moods and your temper and your feelings are not altogether adjusted as you, you are no longer adjusted as you, you are adjusted as a kind of paler version or some diluted form of yourself. You are not quite yourself to receive what's going on... It's a very unusual thing, and I have no way of actually sharing what it is about, as a kind of cultural matter, or at least a subjective matter or a social matter. It's actually fenced off in a kind of limbo state. And that in itself has its own correlation in anguish and isolation."

This is a direct quote from one of the participants of the present study, who has a diagnosis of schizophrenia, describing his experience of depression and persecution. The mention of "psychosomatic intrusion" here refers to painful experiences of physical and psychic attacks, which he believed were perpetrated by a local gang. He described the two principal symptoms resulting from these attacks as "torpor and lassitude". A description like this, of debilitating weariness and lethargy, of having become "a paler version" of oneself, would be recognised by many as a strong indicator of depression. The results of the SCAN interview indicated severe depression, one of the clearest cases in the present participant cohort. This man had never been offered psychological therapy, and had been maintained on a stable dose of a single neuroleptic medication for a number of years. The apparent absence, in this case, of any clinical attempt to address the depressive aspect of the individual's presentation reflects a lack of consensus on best practice (Addington et al., 2002) and a limited understanding of the relationship between depression and psychosis.

It is widely acknowledged that depression is common in people with schizophrenia spectrum disorders, that it is associated with poor functional outcomes and that it should be treated (Siris & Bench, 2003; Buckley et al., 2009; Hausmann & Fleischhacker, 2002). Nevertheless, the proposal that depressive processes might contribute to the occurrence or persistence of positive symptoms like hallucinations and delusions (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Freeman et al., 2002) is relatively radical. Almost all twentieth century accounts of schizophrenia placed depression either as an epiphenomenon of the assumed primary disease process, a side-effect of treatment or, at best, a comorbidity: perhaps resulting from demoralisation – quintessentially secondary. This hierarchy is challenged by evidence suggesting that depression might play a causal, or at least prognostic, role in the trajectory of positive symptoms. If depression were necessarily secondary to schizophrenia, it should not feature prominently in the prodromal phase (Häfner et al., 2005), should not predict transition to psychosis of at-risk individuals (Krabbendam et al., 2005) and should not prospectively predict the severity of positive symptoms, as

demonstrated in the present research and in five previous studies reviewed in Chapter 2. If this pattern of findings is taken to be reliable and valid in reflecting the characteristics of the population of people with schizophrenia spectrum disorders, then it is logical to propose that depression might play a causal role in prolonging or exacerbating psychotic processes, or that common underlying risk factors might drive the association by causing both depression and persisting psychosis.

The theoretical models of Garety, Freeman and colleagues (Garety et al., 2001; Freeman et al., 2002) incorporate causal effects between depression and psychotic symptoms, as well as common vulnerability factors (e.g. negative schematic beliefs) that contribute to their co-occurrence. The assertion that depression can influence psychotic symptoms is not intended to contradict the proposal that psychosis can cause depression (e.g. Birchwood et al., 2000), but the focus here is to address the relative neglect in the literature of the former effect. The evidence of the present investigation concerns persecutory delusions specifically, though other investigators have found support for associations of depression with other psychotic symptoms, such as verbal auditory hallucinations (Soppitt & Birchwood, 1997; Birchwood & Chadwick, 1997).

8.2 Depression and persecutory delusions

At first assessment, it was hypothesised that between 25% and 75% of participants with persecutory delusions would concurrently meet ICD-10 diagnostic criteria for current major depression. This was the usual prevalence range reported from previous studies of depression in schizophrenia by Birchwood et al. (2005). The hypothesis was confirmed in the present study, with a 50% prevalence rate of major depression among participants with persecutory delusions. This is exactly the same figure as the average reported in Buckley et al.'s (2009) review of schizophrenia studies.

8.2.1 Depression predicts the persistence of paranoia

Longitudinally, it was hypothesised that levels of depression would predict the persistence over time of persecutory delusions. This was based on the results of the systematic literature review reported in Chapter 2, which notably included the studies of Drake et al. (2004) and Fowler et al. (2011), both reporting a predictive effect of depression on paranoia. This hypothesis was also confirmed in the present study: for every given baseline paranoia score, higher baseline depression scores significantly predicted higher follow-up paranoia scores. Hierarchical multiple linear regression

modelling indicated that baseline depression scores predicted 10% of variance in follow-up paranoia severity, on top of what could be accounted for by baseline paranoia severity.

The main finding reported here, that depression levels have a predictive effect on subsequent paranoia, seems to be robust, as it parallels the findings of several other studies that used different participant selection criteria, assessment measures and time intervals. Drake et al. (2004) and Fowler et al. (2011) used longer follow-up periods than the present study, 18 months and 12 months respectively. Thewissen et al. (2011) conducted an experience sampling study with measurements taken ten times a day for several days, and also found that depression predicted the severity of subsequent paranoia. The present study sample was mixed in duration of illness, while Drake et al. (2004) used a first episode sample and Fowler et al. (2011) had participants experiencing a relapse. While the present study used the Green et al. Paranoid Thoughts Scales (Green et al., 2008), a questionnaire specially developed to measure paranoia, Drake et al. (2004) and Fowler et al. (2011) extracted paranoia factors from clinical interview assessments, and Thewissen et al. asked participants completing their experience sampling study to rate four items on seven-point Likert scales: 'I feel that others dislike me', 'I feel that others might hurt me', 'I feel suspicious', and 'I feel safe' (reverse scored). One further study reviewed in Chapter 2 (Bergstein et al., 2008) found that depression levels at an initial assessment predicted delusion severity 12 months later, where delusion severity was measured using the Peters et al. Delusions Inventory (Peters, Joseph, & Garety, 1999). The 21-item PDI includes two items with clear persecutory content, and the rest include ideas of reference, grandiosity, control, magical or paranormal beliefs and subjective thought disorder. It is possible that the predictive effect of depression on persecutory ideation might extend to delusions in general, and perhaps to other psychotic symptoms.

8.2.2 The cognitive features of depression accompanying persecutory delusions

Depression occurring in a group of people with persecutory delusions was accompanied by a number of the same cognitive features that characterised a group of people with major depressive disorder, compared to controls. The significance of these cognitive factors goes beyond demonstrating similarity between the comorbid group and those with non-psychotic depression. From a theoretical perspective, certain information-processing biases associated with depression (e.g. memory bias) could contribute to the persistence of persecutory ideation, and other cognitive markers (e.g. negative schematic beliefs) could serve as underlying risk factors for

both affective and psychotic disturbance. As outlined in Chapter 4, the predisposing effects of adverse developmental experiences on later psychopathology are thought to be mediated by the disruption of primary attachments and formation of enduring negative schematic beliefs about the self and others.

8.2.3 Negative schematic beliefs

Negative schematic beliefs have been proposed to predispose individuals to the development and persistence of persecutory delusions (Freeman et al., 2002), as well as depression (Beck, 1976). In the present study, it was hypothesised that among participants with persecutory delusions, those who concurrently met criteria for major depression (PD+D subgroup) would report more negative schematic beliefs than those who were not diagnostically depressed (PD subgroup). Analogously, it was hypothesised that a group with non-psychotic major depression (D group) would show more negative schematic beliefs than a non-clinical (NC) control group. The longitudinal hypothesis was that negative schematic belief scores would predict the persistence of persecutory delusions and depression over time.

The findings confirmed that the PD+D subgroup endorsed significantly more negative beliefs in relation to the self than the PD subgroup. No significant differences were found between the subgroups' schematic belief scores in relation to other people, which may represent a ceiling effect, due to the common presence of persecutory delusions. The D group endorsed significantly more negative beliefs than the NC group on every subscale, as expected.

Analysis of longitudinal data from the combined delusions group confirmed that negative schematic beliefs about the self predicted the persistence of both paranoia and depression over six months. For every given baseline symptom level, more negative schematic beliefs at baseline predicted higher symptom severity at follow-up.

The paranoia prediction result replicates the effect reported by Fowler et al. (2011), over a shorter period of time and with fewer participants. Given that depression and negative schematic beliefs each predicted the persistence of delusions, and that the two predictors were highly intercorrelated, it is likely that depression and negative beliefs predicted overlapping portions of the variance in follow-up paranoia scores. Negative schematic beliefs about the self are a more specific construct than depression, and their role is therefore more straightforward to interpret. The six items of the Self Negative subscale of the Brief Core Schema Scales (BCSS; Fowler et al., 2006) are reproduced in Figure 8.1 to illustrate the content of the measure.

1. I am unloved	NO	/	YES	>	1	2	3	4
2. I am worthless	NO	/	YES	>	1	2	3	4
3. I am weak	NO	/	YES	>	1	2	3	4
4. I am vulnerable	NO	/	YES	>	1	2	3	4
5. I am bad	NO	/	YES	>	1	2	3	4
6. I am a failure	NO	/	YES	>	1	2	3	4

Figure 8.1. The Self Negative subscale of the Brief Core Schema Scales.

Respondents are required first to answer YES or NO. Then, if the answer is YES, a number should be chosen to indicate the strength of agreement with each statement.

The result that negative schematic beliefs about the self also significantly predicted follow-up depression levels, beyond what could be accounted for by baseline depression, suggests that the schematic belief construct has significance beyond playing a symptom role in the depression syndrome. Negative beliefs about the self have been shown to predict the persistence of depression (e.g. Dent & Teasdale, 1988), although no previous study is known to have examined this in a sample selected for the presence of persecutory delusions. Fowler et al.'s (2011) structural equation modelling analysis, seeking to unpick the directions of associations between low mood, negative beliefs about the self and paranoia in people with psychosis, found that the best fitting model had reciprocal effects between low mood and negative beliefs. Negative beliefs, and not low mood, had an additional independent significant effect on paranoia in this model. Causality cannot be inferred from observational research, and it is possible that other unmeasured factors could underpin the effects reported here. However, findings reviewed in Chapter 2 of treatment trials targeting self-esteem in people with psychosis suggest that intervention with self-schematic beliefs can lead to reductions in positive psychotic symptoms as well as in depression (Hall & Tarrier, 2003; Lecomte et al., 1999; Laithwaite et al., 2009; Laithwaite et al., 2007; Knight et al., 2006). This strengthens the case for a causal effect of schematic beliefs on psychopathology.

8.2.4 Responses to distress: experiential avoidance and rumination

Styles of responding to distress, for example with avoidance or rumination, are thought to predispose certain individuals to persistent episodes of emotional disturbance (Nolen-Hoeksema, 1991). The present study findings indicated that depression occurring alongside delusions was accompanied by a similar pattern of

responding to distress as is seen in major depressive disorder. As hypothesised, it was found that the delusions subgroup who met diagnostic criteria for concurrent depression reported significantly higher levels of experiential avoidance and rumination than the subgroup without diagnostic depression. These predicted differences were also found between the depressed and non-clinical control groups, replicating previous findings in the literature.

The longitudinal hypothesis, that these two markers of response style to distress would predict the persistence over time of paranoia and depression, was not supported. Rumination change scores correlated significantly with depression change scores, and experiential avoidance change scores approached a significant correlation with depression change scores, so it would appear that these response style factors were connected with symptom changes, but the lack of evidence for significant predictive effects suggests that they may not have been causally influential.

As mentioned in Chapter 7, the questionnaires used in the present study to measure experiential avoidance and rumination have been criticised for the present purpose (Bond et al., 2011; Ehring & Watkins, 2008; Ehring et al., 2011): both might have less validity for measuring the constructs of interest in people with persecutory delusions than would have been desired. It is possible that other ways of measuring avoidant and ruminative response styles could have enabled detection of predictive effects.

8.2.5 Memory specificity

Reduced specificity of autobiographical memories has been proposed to contribute to the persistence of emotional disturbance by skewing the evidence available for information processing and interfering with problem solving effectiveness (Williams et al., 2007). It was hypothesised that both groups of participants with diagnosable depression – with delusions and without – would show the overgeneral autobiographical memory bias first described by Williams and Broadbent (1986), and that this bias would predict the persistence of paranoia and depression over time.

None of the study hypotheses regarding memory specificity were supported by the results. Instead, the cross-sectional findings of the present study resembled those of Kaney et al. (1999): participants with persecutory delusions, irrespective of subgroup, produced significantly fewer specific memories than each of the groups without psychosis. The longitudinal hypotheses, that AMT performance deficits would predict the persistence of paranoia and depression, also received no support.

The non-replication here of previous findings regarding overgeneral memory in people with depression might be connected with the demographic characteristics of the participant groups, as discussed in Chapter 6. The absence of evidence for significant differences cannot be taken as evidence of the absence of differences, but descriptively the confidence intervals of the depressed and non-clinical groups' AMT scores were mostly overlapping, and the effect size of the difference, if it had been significant, would have been around $d = 0.1$. It is possible that if other factors linked in the literature with memory specificity, such as suicidality and trauma history, had been included in this study, significant associations might have been found.

The unexpected finding that participants with persecutory delusions, regardless of subgroup, performed less well than those without delusions is also interpreted with caution. This finding did not correspond to any *a priori* hypothesis, and might not generalise to the wider population, but it is relevant that Kaney et al. (1999) obtained similar results. The lower AMT performance of the participants with delusions may have reflected a lower general verbal cognitive processing speed or capacity than the participants without psychosis, as indexed by their lower verbal fluency scores and lower premorbid IQ estimates, corresponding to fewer years of formal education and, perhaps, the side-effects of neuroleptic drugs. AMT scores were significantly correlated with IQ estimates, and the correlation between AMT scores and verbal fluency scores approached significance. However, the specificity difference between the participants with delusions and those without remained significant with verbal fluency or IQ estimates controlled in the analysis, so neither of these factors could be sufficient to account for the effect entirely.

The fact that no evidence was found for the prognostic significance of overgeneral memory bias in predicting the persistence of persecutory delusions or concurrent depression over time suggests that Williams' theory of depression persistence (Williams, 2006; Williams et al., 2007) might not be applicable to this population: memory specificity has not demonstrated the same significance here as has been previously found in groups with major depressive disorder.

8.2.6 Problem solving

This was the first study to find that social problem solving performance, measured by the Means-Ends Problem Solving task (MEPS; Platt & Spivack, 1975), could prospectively predict the persistence of symptoms in people with persecutory delusions. Previous findings that problem solving performance predicted prognosis in depression (Garland, Harrington, House, & Scott, 2000) contributed to the

development of a theoretical framework (Williams, 2006) proposing that other associated cognitive features (e.g. avoidance, rumination) exert their depressogenic effects via a common pathway of interference with problem solving, which has an impact on the individual's adaptation to the environment, prolonging affective disturbance. Platt & Spivack's original articles about the MEPS (Platt & Spivack, 1972a; Platt & Spivack, 1972b) also put forward the idea that deficits in social problem solving mediate the maladjusted individual's dysfunction in the world.

At first assessment, the present study findings on problem solving performance were similar to those on memory specificity. The hypothesis that the PD+D subgroup would perform less well on the MEPS than the PD subgroup was not supported, and the D group showed no deficits compared to the NC group. Instead, it was found that participants with delusions, irrespective of depression subgroup, performed significantly less well than those without delusions. In the longitudinal analysis, however, both prospective hypotheses about problem solving performance were supported: low MEPS scores significantly predicted the persistence of both paranoia and depression.

The non-replication of previous findings of problem solving deficits in people with depression was cautiously interpreted, like the AMT findings, with reference to the demographic characteristics of the participant samples. Another study is known to have recruited control participants in a similar way to this one, and similarly did not find a deficit in people with depression, until participants were induced to ruminate (Donaldson & Lam, 2004). The present finding that participants with delusions scored significantly lower on problem solving than those without psychosis fits with their overall lower levels of educational attainment and employment, as well as lower IQ estimates and verbal fluency scores.

Poor social problem solving in people with persecutory delusions is associated with a poor prognosis, both in terms of paranoia persistence and ongoing depression. Problem solving deficits could impede recovery by impairing instrumental behaviours and leading the individual into unhelpful or dangerous situations. Consistent with this proposal, there is an indication in the literature that problem solving therapy can lead to significant symptom improvements for people with psychosis (Tarrier et al., 1993).

8.3 Other cognitive factors, not specifically associated with depression

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) and the Beads Task (Garety et al., 1991) were also included in this study, because previous research had found significant associations of worry and data gathering style with the presence of persecutory delusions. Furthermore, worry has been linked with delusion persistence (Startup et al., 2007). It was of interest how the effects of these processes, if replicated, might fit with the main depression-related features of interest.

8.3.1 Worry

Generalised anxiety disorder is the condition most closely associated with worry, and patients with this disorder tend to score highly on the PSWQ (Chelminski & Zimmerman, 2003). People with persecutory delusions also show high levels of worry (Freeman & Garety, 1999; Morrison & Wells, 2007) - many of them comparable to treatment-seeking patients with generalised anxiety disorder (Startup et al., 2007) - and the same can be true of groups of people with major depression (Starcevic, 1995). The relatively high levels of worry in people with persecutory delusions and those with depression are consistent with high prevalence rates of coexisting anxiety disorders in both groups (Freeman & Garety, 2003; Sartorius, Ustun, Lecrubier, & Wittchen, 1996).

It was hypothesised that all clinical participants in the present study would have significantly higher PSWQ scores at first assessment than the non-clinical control group. However, results indicated that only those with diagnostic levels of depression: the PD+D group and the D group - had significantly higher levels of worry than the NC control group, while the PD group did not differ significantly. This is the same pattern that was observed for rumination: each depressed group scored significantly higher than the corresponding non-depressed group. This could be taken to indicate that the high levels of worry reported previously in people with persecutory delusions could be specific to those with significant levels of concurrent depression. This comorbid subgroup might also experience high levels of anxiety. In the present study, the PD+D group had almost double the average Beck Anxiety Inventory score of the PD group. However, as mentioned in section 8.2.3, it would not be appropriate to draw from the non-significance of a difference between groups the conclusion that they are equivalent. The PD group did report non-significantly higher levels of worry than the NC group, and the difference might have reached significance in a larger participant sample.

No specific longitudinal hypotheses were made in this study regarding worry, but it was included in prospective analyses as a predictor alongside the main depression-related measures. Worry levels at baseline significantly predicted the persistence of persecutory ideation over time, as reported previously (Startup et al., 2007): for every given level of baseline paranoia, higher baseline worry scores were associated with higher follow-up paranoia. This adds to accumulating evidence that worry may contribute to the persistence of persecutory delusions. Two small-scale trials have recently targeted worry in this population, with encouraging results of significant symptom improvement (Foster et al., 2010; Hepworth et al., 2011). A larger-scale RCT is underway, which might extend these findings.

8.3.2 Jumping to conclusions: Beads Task performance

The jumping to conclusions (JTC) data gathering bias that has been previously reported in people with persecutory delusions (Corcoran et al., 2008; Startup et al., 2008) was confirmed in the present sample. It was hypothesised that participants with persecutory delusions would JTC more often than those without delusions, irrespective of depression levels. The results indicated that participants with delusions did JTC significantly more often than those without delusions, in line with previous findings. However, it was unexpectedly found that those among the delusions group who met criteria for concurrent depression showed significantly less JTC than those who did not meet criteria for depression. This is a new finding, was not specifically hypothesised and may not generalise beyond the present sample. Even so, the suggestion of an interaction between affective disturbances and data gathering bias in people with delusions is interesting, as these features have previously been conceptualised as relatively independent (Bentall et al., 2009; Garety et al., 2005).

An additional finding of the present study was that JTC in the delusions group was associated with low problem solving scores on the MEPS. This was an exploratory finding, and might have been due to underlying associations of both factors with IQ. Even so, it is possible that the hasty data gathering style proposed to underlie the JTC phenomenon might also affect the complexity and effectiveness of social problem solving responses.

8.4 Theoretical implications

The present research explicitly set out to extend a multifactorial cognitive-behavioural account of persecutory delusions, described in Chapter 1, with a more detailed

conceptualisation of the role of depression and associated cognitive processes in the maintenance of threat beliefs.

8.4.1 Support and elaboration of a multifactorial model of persecutory delusions

Freeman et al.'s (2002) model implicated both anxiety and depression in driving cognitive and behavioural processes (e.g. attentional and memory biases, avoidant safety behaviours) that might reinforce persecutory delusions. Anxiety and associated processes such as worry and safety behaviours have now received considerable research attention (Freeman & Garety, 1999; Freeman et al., 2001; Freeman et al., 2007; Startup et al., 2007; Foster et al., 2010; Hepworth et al., 2011). The present study findings also support the roles of anxiety and worry in delusion maintenance: both were found to significantly predict the persistence of paranoia over time. The main focus, however, was on processes linked with depression.

Freeman et al.'s account proposed that emotional concerns can drive the content of delusional beliefs directly, and furthermore that the biases in attention and information processing that accompany emotional disorder can impair the reality testing of an established persecutory belief and increase the probability of its persistence. The results of the present research are consistent with this proposal: depression and associated cognitive factors were found to significantly predict the persistence of persecutory ideation over time. Negative schematic beliefs about the self and deficits in social problem solving performance were the cognitive features that showed significant predictive effects. Negative schematic beliefs about the self are specifically mentioned in Freeman et al.'s (2002) model, as well as in Garety et al.'s (2001) broader cognitive model of positive psychotic symptoms, as a factor conferring vulnerability to the development of delusions. Previous studies have reported that such beliefs can predict subsequent paranoia (Drake et al., 2004; Fowler et al., 2011), and the present findings accrue to support the robustness of this effect. The finding that deficits in problem solving predict the persistence of persecutory ideation is new, and fits into Freeman et al.'s (2002) model of delusion persistence alongside other cognitive features that increase the probability of the individual obtaining biased evidence to support the delusional threat belief.

8.4.2 Problem solving difficulties might mediate the effects of other factors

The role of problem solving deficits in psychopathology has been conceptualised, albeit not with specific reference to delusions, as a common process mediating the individual's adaptation to the environment, by interference with which other more simple disturbances might exert their effects on symptom persistence (e.g. Platt & Spivack, 1972a; Williams, 2006). If the role of problem solving deficits in delusion persistence is conceptualised in this way, it could be proposed that more specific cognitive biases such as JTC might contribute to delusion persistence by interfering with problem solving effectiveness. The present findings suggested an association between JTC and problem solving performance (although it may have been explained in this case by bilateral associations with IQ). It would be premature to claim a role for problem solving performance in mediating the effects of other processes on delusion persistence before carrying out formal mediation analysis. Nevertheless, this is a plausible and empirically supported component in the cycle of symptom maintenance.

8.4.3 Reflections on other theoretical accounts of the relationship between delusions and depression

Several other conceptualisations of the relationship between delusions and depression were outlined in Chapter 2, notably Birchwood and colleagues' social ranking theory account of post-psychotic depression (Birchwood et al., 1993; Birchwood et al., 2000) and Bentall and colleagues' attribution-focused defence model of persecutory delusions (Bentall et al., 1994; Bentall et al., 2001). The present study was not designed to test specific predictions arising from either of these accounts, but results regarding schematic beliefs and avoidance have some relevance.

The finding that among people with persecutory delusions, negative schematic beliefs about the self are more prevalent in those with concurrent depression is consistent with Birchwood et al.'s proposal that depression arises in people with psychosis as a result of the detrimental impact of the content and meaning of the psychotic experience on the individual's subjectively perceived value or social standing. Moreover, negative beliefs about the self were found to predict the persistence of depression in this group, which would have been predicted by Birchwood et al.'s model. However, no test is available here of whether or not the psychotic experience contributed to these negative self-beliefs, which might have existed before the onset of the disorder.

Bentall and colleagues' proposal that problems of self-esteem lie at the heart of persecutory delusions is also consistent with the evidence reported here concerning the role of schematic self-beliefs. Perspective is limited on the more controversial 'defence' aspects of Bentall and colleagues' theory, because the present study did not include any measures of covert vs. overt self esteem or of attributional style. Perhaps if defensive processes were supposed to contribute to the genesis of persecutory delusions by protecting the self from covert negative self-appraisals, it might have been expected that reports of self-opinion on the BCSS questionnaire (overt) would not have shown a direct relationship with the course of paranoia. The proposal that paranoia can be subdivided into two types (Trower & Chadwick, 1995): a defensive 'poor me' type and a non-defensive 'bad me' type – also cannot be evaluated against the present findings, as no measure of deservedness was included. It is possible that the PD subgroup (no diagnostic depression) would roughly correspond to the 'poor me' defensive type, and the PD+D subgroup (meeting criteria for depression) to the 'bad me' non-defensive type, following Trower & Chadwick's (1995) description of the two typical presentations. The instrument included in the present study most akin to something measuring a defensive process as described by Bentall and colleagues would be the Acceptance and Action Questionnaire, measuring experiential avoidance. The fact that avoidance was significantly higher in the PD+D group than the PD group would not be consistent with a proposal that non-depressed people with delusions are engaged in a defensive process of avoiding contact with underlying negative self-evaluations. No direct evidence has been gathered in the present study to evaluate the relative validity of a defensive model of persecutory delusions. It can, however, be said that the present pattern of findings is most closely in keeping with Freeman et al.'s (2002) theory of direct, non-defensive effects.

8.5 Clinical implications

The best approach to helping people with concurrent depression and psychosis is not known. An international survey of 80 000 consultant psychiatrists (Addington et al., 2002) found that despite widespread acknowledgement of the prevalence and burden of depression in people with schizophrenia, there was little agreement on the best treatment strategy. Addington and colleagues suggest that the lack of consensus on management reflects clinicians' differing beliefs about the ontological status of the depressive symptoms observed. For example, if seen as an inherent part of the psychosis symptom profile, depression might be expected to respond to antipsychotic drugs, whereas if the clinician perceives a 'superimposed' depressive episode, antidepressants might appear appropriate. The most common treatment approach reported by respondents of the survey was augmenting neuroleptics with

antidepressant medications, a practice for which empirical support is weak (Whitehead, Moss, Cardno, & Lewis, 2003; Micallef, Fakra, & Blin, 2006).

Psychological therapies, including CBT, have demonstrated concurrent efficacy in the reduction of depression and psychotic symptoms, which suggests that this is a good treatment option for people experiencing psychosis with depression. In Chapter 2, the findings were systematically reviewed of treatment trials that reported significantly reducing depression in people with schizophrenia spectrum disorders. Of these, 12 were medication trials and 14 were trials of psychological therapy. Four of 12 medication trials and 11 of 14 psychological therapy trials reported therapeutic benefits for psychotic symptoms as well as for depression. Psychological therapies such as CBT usually involve the development of an idiosyncratic formulation that makes sense of the individual's presenting set of difficulties, upon which treatment is then based. This might make it conceptually easier for practitioners of psychological therapies to deal clinically with comorbidity, especially as psychological models of various disorders are often adapted from one to another, while pharmacists and prescribers must contend with the interactions of chemically diverse drug compounds.

8.5.1 Depression-related interventions for people with psychosis

Indications that depression, negative self-schematic beliefs and problem solving difficulties might contribute to the persistence of persecutory delusions in people with psychosis suggest that targeting these factors therapeutically might improve the recovery of affected individuals. CBT for psychosis was adapted from Beck's original protocol targeting depression (Beck, Rush, Shaw, & Emery, 1979), and the diagnosis-differentiated packages continue to share a number of techniques, such as exploring alternative interpretations of experience and targeting negative core beliefs (Morrison & Barratt, 2010). Given that 14 psychological therapy trials reviewed in Chapter 2 significantly reduced participants' depression levels, it is likely that depression is already targeted to some degree in the psychological therapies for psychosis that are currently in use. The results of the present study support the inclusion of depression as an explicit treatment target in these therapies.

Reports of the efficacy of self-esteem interventions for people with psychosis support the utility of addressing negative beliefs about the self (Lecomte et al., 1999; Laithwaite et al., 2007; Hall & Tarrier, 2003; Knight et al., 2006). The results of the present study, in line with Fowler et al.'s (2011) findings, suggest specifically that high levels of *negative* schematic beliefs about the self are predictive of symptom persistence, not low levels of positive beliefs. The clinical implication is that reducing

negative beliefs about the self might be a more specific and helpful therapeutic target than increasing positive beliefs. Most self esteem therapy packages focus on building positive beliefs (e.g. Hall & Tarrier, 2003; Laithwaite et al., 2007; Lecomte et al., 1999), and fewer explicitly tackle negative beliefs (e.g. Laithwaite et al., 2007). It is likely that increasing positive beliefs about the self has an indirect effect on negative beliefs, but the implication of the present findings is that explicitly targeting negative beliefs might bring about symptom benefits more directly. One example of a psychological intervention that might achieve therapeutic effects by breaking down negative self-evaluations is Compassionate Mind Training (Gilbert & Procter, 2006). This was developed specially to address self-attacking and self-blame in individuals with long-standing mental health problems, who might be affected by drastically disturbed attachment experiences. A compassion-focussed intervention has demonstrated significant beneficial effects on depression levels and self-esteem in a group of high-security inpatients with psychotic disorders (Laithwaite et al., 2009). No significant changes in positive symptoms were found, but this may represent a floor effect, as positive symptoms were minimal on entry to the trial.

Not all empirically-supported depression treatments have been investigated in application to psychosis. For example, behavioural activation (BA), whereby activity scheduling is used to increase the individual's exposure to rewarding stimuli in order to lift mood, has well-established efficacy in the treatment of depression (Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008; Mazzucchelli, Kane, & Rees, 2009), and may be just as effective as more complex CBT interventions (Jacobson et al., 1996). A pilot study trialling BA for psychosis (Mairs, Lovell, Campbell, & Keeley, 2011) reported promising results from 8 patients indicating feasibility, treatment adherence and initial outcomes: non-significant improvements in depression, positive symptoms and negative symptoms. No controlled trials are yet known to have been published of BA for people with psychosis. Given the body of evidence for the therapeutic effect of this approach for major depressive disorder, trialling BA as an intervention for comorbid depression in people with psychosis could also be of benefit.

8.5.2 Problem solving training

Of the cognitive factors showing significant predictive effects in the present investigation, problem solving appears to have received the least interventionist empirical attention in relation to delusions, with one notable exception. Tarrier et al. (1993) implemented a problem solving intervention as part of an early comparative trial of cognitive behavioural treatments for people with drug-resistant psychosis. The

main intervention being investigated here was Coping Strategy Enhancement (CSE; Tarrier, Harwood, Yusopoff, Beckett, & Baker, 1990); problem solving (PS) was chosen for the active control condition, as "a credible treatment which may well result in benefits to the patient but would not be expected to directly address psychotic symptoms." The six-week PS intervention was based on D'Zurilla & Goldfried's (1971) formulation of problem solving. Participants were encouraged to identify their current personal problems, and then they were trained in an iterative systematic process of alternative solution generation, selection, implementation, evaluation and self-reinforcement. After training, the strategy was applied to abstract problems such as board games, then to realistic social scenarios like making friends or finding a job. Finally, the systematic problem solving approach was applied to participants' own previously generated personal problems. CSE, unlike PS, specifically concerned the self-management of residual psychotic symptoms. It was predicted that CSE would improve psychotic symptoms and also thereby have a secondary effect on social functioning, while PS would only improve social functioning without changing symptom levels. On the contrary, both interventions had the effect of improving psychotic symptoms (which did not change over a waiting list control period), but no significant changes in social functioning were observed under either condition. Specific treatment effects were observed on delusions, as well as anxiety. Symptom improvements were greater in the CSE group, but this group had entered the trial (as an unintended effect of randomisation) with a mean total symptom score more than double the PS group's, which makes the differential changes difficult to interpret. Nevertheless, the suggestion that these two types of psychological therapy led to significant improvements in medication-resistant delusions is intriguing.

An earlier study (Falloon et al., 1985) had also used an individual problem solving intervention as a control treatment for a group with psychosis, this time against family CBT. Here, like in Tarrier et al.'s (1993) study, the emphasis on the superiority of the more complex intervention overshadowed the apparent benefits of problem solving training. Problem solving has continued in use as a component of cognitive behavioural interventions for psychosis, but received little further attention as a stand-alone treatment for some time, until a resurgence of interest by researchers in Italy. Falloon and colleagues (Falloon, Barbieri, Boggian, & Lamonaca, 2007) laid out a theoretical framework of pathways by which PS might benefit people with psychosis, which includes stress management, increasing interpersonal competence, containing emotional hypersensitivity, remediating neurocognitive deficits and even functionally restructuring neural circuitry. The same article presents a summary of promising findings from four pilot studies, which showed improvements in positive symptoms,

negative symptoms, cognitive and occupational functioning of people with psychosis who completed problem solving interventions. A problem solving training (PST) course is described, based on the same core systematic problem solving technique taught by Tarrier et al. (1993), but delivered in a group format, extended to 12 months and incorporating worksheets and guidebooks, in a deliberate analogy to the mainstream teaching of mathematics or foreign languages. A multicenter randomised controlled trial of this training programme is said to be underway, which aims to include 120 patients. This would be the largest known study of problem solving therapy for people with psychosis.

Tarrier et al.'s (1993) study is the only one known to have examined the effects of PS on delusions specifically, and it is encouraging that positive findings were obtained with an intervention as short as six weeks, but replication will be necessary to demonstrate the reliability of any such effects. No other studies are known to have examined the effects of PS on individual positive symptoms, perhaps because the content of the intervention does not immediately appear relevant to symptom reduction. The present study's finding that problem solving deficits predict the persistence of persecutory delusions in people with psychosis is certainly consistent with a causal role for problem solving skill in the resolution of psychotic symptoms, and the allied possibility that problem solving training can create "some kind of cognitive change that inhibits delusions or hallucinations" (Tarrier et al., 1993, p. 530). An analogy can be drawn with interventions targeting worry (Foster et al., 2010) self esteem (Hall & Tarrier, 2003; Knight, Wykes, & Hayward, 2006; Laithwaite et al., 2007) and insomnia (Myers et al., 2011), which have also achieved reductions in positive psychotic symptoms without challenging these symptoms directly. The accessibility and face validity, from the point of view of the participant, of problem solving training add to its appeal as a cognitive treatment approach, and its potential efficacy in treating positive symptoms such as persecutory delusions merits further exploration.

8.6 *Limitations of the present investigation*

The present investigation set out to examine the case for a role of depression in the persistence of paranoia, and efforts were made to measure and account for a range of relevant factors from the literature on depression and on persecutory delusions. The selection of study measures was theory-driven, and based on instruments used in previous studies, so that the findings might be comparable. Nevertheless, some of the measures yielded inconsistent findings, and the design of the investigation was necessarily associated with some limitations.

8.6.1 The scope of the investigation

A focus on the hypothesised effect of depression on persecutory delusions, and on theory-driven hypothesis testing, precluded the examination of other factors and causal connections that might have been relevant. For example, the primary finding of the present study concerned depression predicting the persistence of paranoia, but the causes of this depression were not addressed. If depression accompanying psychosis can represent several distinct aetiologies (Birchwood, 2003), then the trajectories and consequences of these cases might also separate into groups with different profiles. The present study concerned the prospective main effect of depression, and aetiology was not investigated. The inclusion of more measures might have allowed further theoretical implications to be examined, as mentioned in section 8.4.3, but it was deemed important to limit the duration of the assessments in order not to overload participants who might have difficulty concentrating for extended periods of time.

A basis in multifactorial theory drove the investigation of a number of cognitive factors whose involvement was of interest, and, correspondingly, a number of statistical tests were carried out on data gathered from the same participants, which can increase the study-wide probability of Type 1 error occurring. To mitigate this risk, omnibus tests like MANOVA were used where possible. These aim to control the probability of Type 1 error occurring by adopting a conservative null hypothesis rejection criterion, scaled by the number of possible comparisons. The study analyses emphasised testing one primary cross-sectional hypothesis and one primary longitudinal hypothesis, and auxiliary tests were used to help contextualise the findings alongside those of previously published research.

8.6.2 The role of anxiety

The results of the present investigation indicated that anxiety, as well as depression, was a significant predictor of the persistence of paranoia over time. This is in line with the findings of previous research (e.g. Startup et al., 2007), and with Freeman et al.'s (2002) model. Anxiety was not controlled for in the longitudinal regression analyses, because it was expected that this could obscure the significant effect of depression, with which anxiety shows considerable overlap (Kaufman & Charney, 2000; Brady & Kendall, 1992). The present study was not powered to detect a predictive effect of depression on paranoia persistence while controlling for anxiety, and the present results do not indicate whether or not depression is still a significant predictor when

the effect of anxiety is taken into account. This could be examined in future research with a greater number of participants.

8.6.3 Measurement of experiential avoidance using the Acceptance and Action Questionnaire (AAQ)

This instrument, included in Appendix C, has been criticised for having poor comprehensibility and psychometric properties (Bond et al., 2011). None of the items explicitly mention avoidance or acceptance, and some were difficult to understand for some of the participants of the present study, for example item 6: "When I evaluate something negatively, I usually recognize that this is just a reaction, not an objective fact." Bond et al. (2011) suggested that the AAQ, which has often been used in the context of Acceptance and Commitment Therapy (ACT), may not be adequately comprehensible to people not exposed to contextual CBT approaches such as ACT. These authors also highlighted that particularly poor reliability has been reported when the AAQ has been used with people who are not very highly educated or who have English as a second language. Both of these were true for a considerable proportion of the present study participants. It is possible that a different approach to measuring avoidance could yield significant findings in this population where the AAQ has not.

8.6.4 Ambiguous group differences in problem solving performance

A novel finding of the present study concerned problem solving performance as a predictor of symptom persistence. Poor performance on the MEPS significantly predicted the persistence of both paranoia and depression over time, but the cross-sectional group comparison results were unexpected. It had been hypothesised that deficits in problem solving would be associated with both depression and delusions, and that the group with both types of symptom would perform worst of all. Instead, both groups with delusions demonstrated equivalent deficits in comparison to those without delusions. In this sense, problem solving performance did not behave as a "depression-related" factor in this group, as it had been originally conceived. It was never intended that the depression-related cognitive factors included in this study be thought of as exclusively associated with depression, but the absence of an association with depression makes it harder to interpret what MEPS deficits might serve as a marker of, or which other patient groups might be expected to demonstrate them. The participants with delusions in the present study had significantly lower MEPS scores than those without delusions, but this cannot be said to indicate that

MEPS performance is associated with delusions, because there were a number of other uncontrolled differences confounding the comparison. The delusions group differed from those without delusions not only on the basis of psychotic symptoms, but also in demographic characteristics, IQ estimates and verbal fluency.

8.6.5 Socio-demographic group differences

The demographic characteristics of the participant groups were not matched, and significant differences were evident in ethnicity, employment status and levels of education. Efforts had been made to select representative samples of each population. Socio-demographic disadvantages are thought to characterise populations with psychosis compared to those without (e.g. Kendler, Gallagher, Abelson, & Kessler, 1996), and the present study samples reflected this. It might have been possible to artificially select samples matched for certain demographic characteristics, and thus control for these in a comparative analysis, but the participant samples would have given skewed representations of the characteristics of the broader populations of interest, reducing the generalisability of the findings.

8.6.6 Limitations of the participant sample and observational design

All clinical participants of the study were outpatients, so that more severe acute presentations of psychosis may have been under-represented. The recruitment materials described the aims of the study, so even though the psychosis group was selected for persecutory delusions irrespective of depression, the study description might have skewed referring clinicians to suggest individuals who were depressed. Efforts were made to follow up individuals in the delusions group six months after first assessment, but participants were not always available when contacted, and in one extreme case the second assessment took place 12 months after the first. If any conclusions were to be drawn about the time-frame of predictive effects, the heterogeneity of follow-up intervals should be taken into account.

Observational research is ultimately unable to provide evidence of the directions of causal associations between constructs. The longitudinal design of the present study allowed some evidence of temporal relationships to be gathered, which is a step in the right direction. For example, the finding that for every given level of baseline paranoia, poorer baseline problem solving performance predicted higher follow-up paranoia constitutes stronger evidence for a causal effect of problem solving on paranoia persistence than a cross-sectional correlation could have provided, but it is still

possible that a third factor might have influenced both predictor and outcome. Manipulating variables in a randomised design can help to demonstrate causal relationships. An interventionist-causal model (Freeman, 2011; Kendler & Campbell, 2009) suggests a pragmatic research framework where each individual putative causal factor, once identified, can be manipulated therapeutically. If symptom benefit is demonstrated relative to a control condition, then sufficient causality is said to have been established for practical purposes.

8.7 Directions for future research

The limitations of the present results can be addressed through replication in other samples and triangulation using different methodological approaches. This study was not the first to find that negative schematic beliefs and worry predict the persistence of persecutory beliefs (Fowler et al., 2011; Startup et al., 2007), and intervention studies targeting these factors have already shown beneficial effects on delusions (Laithwaite et al., 2007; Foster et al., 2010). It may be possible to trace the origin of these unhelpful cognitive processes further back, for example to traumatic life experiences and disrupted early attachments. A reliable association has been indicated by several reviews of the literature between psychosis and trauma, particularly childhood trauma (Varese et al., 2012; Read, Van Os, Morrison, & Ross, 2005; Morrison, Frame, & Larkin, 2003). High rates of insecure attachment styles have also been reported in people with psychosis (e.g. Couture et al., 2007; Ponizovsky, Nechamkin, & Rosca, 2007; MacBeth, Gumley, Schwannauer, & Fisher, 2011). Investigation of the mechanisms, such as attachment and affect regulation, by which early adversity can lead to psychiatric symptoms, will bring us closer to a complete psychological understanding of psychosis.

It might also be possible to clarify, in future studies with more sophisticated measures, the inconsistent effects found here for certain cognitive processes. For example, the present results do not suggest a relationship between memory specificity and delusion persistence, but future investigations might obtain more interesting results if participants' trauma history and any post-traumatic symptoms are assessed, as these have shown a relationship with AMT performance (Williams et al., 2007).

8.7.1 Replication and elaboration of problem solving effects

The finding here that social problem solving deficits predict the persistence of persecutory delusions and depression in people with psychosis is novel and intriguing, but, because of its novelty and the context of multiple testing, must be taken as

preliminary. It would be of value to replicate this effect, because if it could be established reliably that problem solving deficits predict the persistence of symptoms then immediate clinical implications could be addressed, as discussed in section 8.5.2.

The significant correlations of problem solving performance with verbal fluency and IQ estimates highlight the associations of 'higher' cognitive performance parameters, like social problem solving, with more general characteristics of processing capacity, speed or even vocabulary. No comprehensive neuropsychological examination was included in the present protocol, and the basic cognitive measures used – the WTAR and the verbal fluency test – can say little about the qualitative nature of any deficits recorded. Neuropsychological assessment batteries can be highly taxing and time-consuming: unfeasible for the present study, which aimed to look at the interlocking effects of a broad array of measures in a sample of people with psychosis who often experience difficulties concentrating for extended periods of time. Given that social problem solving performance has here demonstrated significant associations with symptom persistence and with other cognitive markers, it might ultimately be desirable to break down the effect of this construct into simpler components. There may be certain relevant aspects of cognition which are more amenable to training than others, and some that may have particularly strong links with symptom prognosis. From a clinical point of view, a more nuanced understanding of the roles of problem solving components could enable optimally helpful therapeutic strategies to be developed.

8.7.2 Negative repetitive thinking

It is thought that rumination and worry share the same process of negative repetitive thought (Segerstrom et al., 2000; Watkins et al., 2005), differing in content according to their principal associations with depression and anxiety, respectively (McLaughlin et al., 2007). Ehring and Watkins (2008) proposed that negative repetitive thinking is a transdiagnostic process that perpetuates a large range of Axis 1 disorders.

In the present study, worry significantly predicted the persistence of paranoia over time, as found previously (Startup et al., 2007), but the predictive effect of rumination did not reach significance. Worry has been argued to maintain persecutory delusions by keeping the belief content in mind through repetition and by impeding full emotional processing of the content (Freeman, 2007; Hepworth et al., 2011). It would follow that rumination, as another form of negative repetitive thought, should have a similar effect. The present differential findings suggest that some kinds of negative repetitive thought might be more strongly associated with symptom persistence than others.

Further gains in disentangling the relative effects of the process and the content of negative repetitive thinking might be possible with the use of measures developed to be content-independent (e.g. Ehling et al., 2011).

8.7.3 Psychotic symptoms beyond persecutory delusions

The present investigation focused on persecutory delusions as a very common and debilitating psychotic symptom (Sartorius, Jablensky, & Korten, 1986; Appelbaum et al., 1999), but depression may be involved in the persistence of different delusion subtypes and other psychotic symptoms. Depression is likely to have different relationships with various psychotic phenomena, such as different delusion types (e.g. Garety et al., 2012), which systematic investigation could reveal. Cross-sectional associations have been found of depression with aspects of hallucinated voices such as malevolence, derogatory content and intrusiveness (Soppitt & Birchwood, 1997; Birchwood & Chadwick, 1997). An integrated theory of psychosis would benefit from an understanding of how emotional disturbance can contribute to the range of different hallucinations and delusions.

8.7.4 Other diagnostic categories

The present investigation selected samples of diagnostically-defined participants with schizophrenia-spectrum disorders and with non-psychotic major depressive disorder, in order that the findings would be comparable to those of previous research using the same standard diagnostic criteria. However, the symptoms of interest – persecutory delusions and the symptoms of depression – do not occur exclusively in these well-defined groups, and it will be important to understand to what extent the same effects apply to each symptom transdiagnostically. People with persecutory beliefs in the context of psychotic depression, and those with bipolar disorders, would be of particular interest.

8.8 Overall conclusion

Depression is common in people with persecutory delusions, and is associated with a number of cognitive features seen in major depressive disorder. Levels of depression predict the persistence of persecutory delusions over six months. Evidence of depression-related cognitive features in people whose low mood accompanies a schizophrenia-spectrum disorder suggests mechanisms that might connect emotional disturbance with psychosis.

Negative schematic beliefs about the self and social problem solving difficulties predict the persistence of both persecutory delusions and depression over time. The finding that shared prognostic processes significantly predict the persistence of both depression and paranoia provides empirical support for the application of similar therapeutic approaches to the treatment of these problems. The present findings support the relevance of current clinical research trials targeting beliefs about the self and worry in people with delusions. Translation of further techniques from depression treatment for use with people experiencing psychosis may be indicated.

The significance that negative beliefs about the self can have for the manifestation of psychotic experiences is illustrated by the following quote, from a man who wanted to participate in this study, but was excluded because he no longer held a persecutory delusion. He had previously believed that members of the community, who could all read his mind, were attacking him psychically and exerting control over his actions. Over the course of treatment, which included a course of CBT, his beliefs about his own faultiness and vulnerability were identified and addressed, and incrementally he came to think less negatively of himself. By the time that he volunteered to take part in this study, the attacks had completely stopped, because the people – who nevertheless could still read his mind – could tell that he was no longer an easy target.

"[I was] vulnerable, confused, lost, scared, frightened. People could see what stage I was at. I felt I was like an open book. Everyone around me could hear what I was thinking about in my mind. They wanted me to be a sex slave... Looking back now, it used to worry me so much back then, but it doesn't bother me now, because people know they can't do that now. They don't even try to do that now, because they roughly know what stage I'm at, and they know that there's no way they can take control of me now, because they know I'm my own boss, and I'm doing my own thing."

It is hoped that the work presented in this thesis will contribute to the further development of understanding and treatment for people who have similar experiences.

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Appendices

Appendix A. Ethical approval letters

Moorfields & Whittington Research Ethics Committee

Royal Free Hospital
Pond Street
London
NW3 2QG

Telephone: 020 7794 0552

Miss Natasha Vorontsova
PhD student
Institute of Psychiatry, King's College London
Department of Psychology, PO Box 78
ASB, 4 Windsor Walk, Institute of
Psychiatry, King's College London
SE5 8AF

04 November 2009

Dear Miss Vorontsova

Study Title:	Cognitive factors maintaining persecutory delusions in psychosis: the contribution of depression.
REC reference number:	09/H0721/61
Protocol number:	1

The Research Ethics Committee reviewed the above application at the meeting held on 28 October 2009. Thank you for attending to discuss the study.

Ethical opinion

The Committee welcomed the opportunity to ask you further clarification on the following points:

There should be a GP letter informing that the participants are taking part in the study.

You asked whether the GPs for the non-clinical control group should be informed as well and the Committee confirmed that would not be necessary.

In the section, 'Why have I been invited to take part?' in the Delusions Group and the Depression Group Information Sheets, the Committee thought that the wording, 'we have been told...' suggests that confidential issues have been discussed between the clinical team and the researchers. It would be better to rephrase the sentence, 'you may have worries' (Delusions Group) or 'you may be experiencing depression' (Depression Group).

You stated that should be possible. You said that you would only ask for permission of the consultants of the clinical teams to approach patients within their care and then ask the clinical team how to approach suitable patients.

How long will the participants have to decide and how will they be approached?

You said that the study would be discussed with the participants and the Information Sheet would be given to them. They would be given at least 24 hours to make a decision before she called to see if they were interested.

It was noted that only participants with persecutory delusions would complete the assessments of the severity of their symptoms twice, once with the initial assessments and then six months later. The Committee thought that all the groups could be assessed a second time to see if there were any changes with the other groups as well. This could provide a baseline and check on the repeatability of the measures.

You said that the aim of the study would be to test the hypothesis that cognitive features predict delusion and so only the delusion groups (with or without depression) would be followed up. You also said that she did not think it would be worth following up each group.

Will you be asking what treatment the participants will be on?

You said that the participants will give consent for her to access their medical records to check diagnosis and medical treatment.

It was noted that in the Participant Information Sheet for the Delusions Group, the last two paragraphs in the section, 'What will happen to the information I give in the study?' did not appear in the other Information Sheets. The Committee thought that the last paragraph (should the participant lose capacity during the study) should appear in all the Information Sheets and the second from last paragraph (sharing important information with clinical team) should also be in the Information Sheet for the Depression Group but not in the Non-clinical Controls Information Sheet.

You said that was a good point.

We noted that one Peer Review asks how you would control for the effects of treatment over time. How would you propose to do this?

You said that she did not think she would be able to do this. If a difference became apparent she would follow it up.

There are quite a lot of valid questionnaires being used. Have they been validated to use together?

You stated that there are two clusters of measures being used. The questionnaires investigating delusion and psychosis have been used together and so have the depression measures been used together.

Will there be anyone else with you when you conduct the interviews and what would you do if someone did get upset during the interviews?

You said that she would be sensitive to participants who may become upset and allow breaks if necessary. All the interviews will be conducted on the NHS site or the Institute of Psychiatry where your supervisors or clinical psychologists would be available if the need arose.

Do you expect to see a change in the cognitive behaviour of participants with delusions?

You stated that these patients are relatively stable. Hopefully the sample size should be large enough to show any change.

We noted that should undiagnosed depression be revealed, the researcher would seek the participant's permission to inform the clinical team. We thought that permission should not be sought from the participant as it would be better if the clinician was told so that the patient could be treated for depression as soon as possible if necessary. This could be made more explicit in the Information Sheets and Consent Form and if a patient does not agree to the clinician being informed, they should not be included in the study.

You agreed to the statement.

There may be some difficulties in distinguishing between feelings caused by depression or some negative schizophrenic feelings such as lack of motivation. How would you deal with this problem?

You agreed that distinguishing between the two conditions could be difficult, but the clinicians would have already made the diagnosis.

One of the Peer Reviews has suggested that you should recruit more participants. Has this been done?

You said that the numbers of participants has been increased in response to the Peer Review.

Ethical Opinion

Decision: Favourable Opinion (with additional conditions)

The members of the Committee present gave a favourable ethical opinion (with additional conditions) of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

For NHS research sites only, management permission for research ("R&D approval") should be obtained from the relevant care organisation(s) in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>. Where the only involvement of the NHS organisation is as a Participant Identification Centre, management permission for research is not required but the R&D office should be notified of the study. Guidance should be sought from the R&D office where necessary.

Sponsors are not required to notify the Committee of approvals from host organisations.

Other conditions specified by the REC. Final versions of documents should be provided to the committee for information, e.g. information sheet

1. Please include a GP letter for the patient groups. This should explain that one of the GP's patients has agreed to take part and inform of the aims of the study and the patient's involvement.
2. There should be a section in the Confidentiality sections of the Delusion's and Depression's Group Information Sheets informing the participant that the GP will be informed.
3. Please ensure that the second from last paragraph in the section, 'What will happen to the information I give in the study?' in the Delusions group Information Sheet, is also included in the Depression Group Information Sheet. Also, the last paragraph of the same section should be included in both the Depression Group and Non-Clinical Group Information Sheets.
4. In the section, 'Why have I been invited to take part?' of both the Delusions Group and the Depression Group Information Sheets, the Committee thought that the wording, 'we have been told...' should be rephrased to 'you may have worries' (Delusion Group) or 'you may be experiencing depression' (Depression Group).
5. There should be two Consent Forms, one for the Patient Groups and one for the Non-clinical Group. All statements (even 1-3) should have boxes for the participant to acknowledge. Please see Guidance on Patient Information Sheets and Consent forms on the website www.nres.npsa.nhs.uk
6. The Consent Form for the Patient Groups should have a section stating that the participant agrees to the GP being informed of their participation in the study.
7. Also in the Consent Form for the Patient Groups, there should be a section stating that the participant agrees to the clinical team being informed of important medical information that may arise during the study.
8. Both Consent Forms should have a statement, 'I understand that relevant sections of my (medical notes and) data collected during the study, may be looked at by individuals from the research team, regulatory authorities, or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records. In the Non-clinical Consent Form, the words, 'medical notes' may not be necessary.

The REC nominated the Co-ordinator, Mrs K Clark, to be point of contact should further clarification be sought from the applicant upon receipt of the decision letter.

It is responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The documents reviewed and approved at the meeting were:

<i>Document</i>	<i>Version</i>	<i>Date</i>
Covering Letter		30 September 2009
REC application	1	30 September 2009
Protocol	1	30 September 2009
Investigator CV		30 September 2009
Participant Information Sheet: Delusions Group	1	30 September 2009
Participant Information Sheet: Depression Group	1	30 September 2009
Participant Information Sheet: Non Clinical Group	1	30 September 2009
Participant Consent Form	1	30 September 2009
Evidence of insurance or indemnity		31 July 2009
Summary/Synopsis	1	30 September 2009
Questionnaire: Validated: Paranoid Thoughts Scale		
Questionnaire: Validated: Beck Depression Inventory		
Questionnaire: Validated: Beck Anxiety Inventory		
Questionnaire: Validated: Brief Core Schema Scales		
Questionnaire: Validated: Ruminative Response Scale		
Advertisement	1	30 September 2009
Two Supervisor CVs		30 September 2009
Four Referee' or Other Scientific Reports		
Scan Depression Schedule	1	30 September 2009
PSYRATS Delusions Scale	1	30 September 2009
Questionnaire: Validated: Penn State Worry		
Questionnaire: Validated: Avoidance and Acceptance		
Questionnaire: Non - Validated: Demographic Information Form		
Questionnaire: Non - Validated: Visual Analogue Scales of Mood		

Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document “After ethical review – guidance for researchers” gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

09/H0721/61	Please quote this number on all correspondence
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With the Committee's best wishes for the success of this project

Yours sincerely

Dr David Slovick
Chair

Email: katherine.clark@royalfree.nhs.uk

Enclosures: List of names and professions of members who were present at the meeting and those who submitted written comments
“After ethical review – guidance for researchers”

Copy to: Jennifer Liebscher, King's College London, R&D office

Moorfields & Whittington Research Ethics Committee**Attendance at Committee meeting on 28 October 2009****Committee Members:**

<i>Name</i>	<i>Profession</i>	<i>Present</i>	<i>Notes</i>
Dr Waheeb Atia	Retired Consultant	Yes	
Mr David Barr	Retired Magistrate	Yes	
Ms Jill Bloom	Drug Information Pharmacist	Yes	
Dr Elizabeth Carrey	MSc Programme Director in Clinical Paediatrics	Yes	
Mr Dan Ehrlich	Head of Optometry	Yes	
Mrs Ros Goldfarb	Retired Immigration Judge	No	
Mr Robert Goldstein	Retired Economist	Yes	
Mr Hari Jayaram	Clinical Scientist in Ophthalmology	Yes	
Dr Stella Kingett	Consultant Psychiatrist	Yes	
Professor Diana Kornbrot	Professor of Mathematical Psychology	Yes	
Ms Mary Ryan	Personnel Manager	Yes	
Dr David Slovic	Consultant Physician	Yes	

Also in attendance:

<i>Name</i>	<i>Position (or reason for attending)</i>
Mrs Kathy Clark	REC Co-ordinator
Mr Tom Lucas	Senior Research Ethics Service Manager

Moorfields & Whittington Research Ethics Committee

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06 November 2009

Miss Natasha Vorontsova
 PhD student
 Institute of Psychiatry, King's College London
 Department of Psychology, PO Box 78
 ASB, 4 Windsor Walk, Institute of
 Psychiatry, King's College London
 SE5 8AF

Dear Miss Vorontsova

Full title of study: Cognitive factors maintaining persecutory delusions in psychosis: the contribution of depression.
REC reference number: 09/H0721/61
Protocol number: 1

Thank you for your email of 06 November 2009. I can confirm the REC has received the documents listed below as evidence of compliance with the approval conditions detailed in our letter dated 28 October 2009. Please note these documents are for information only and have not been reviewed by the committee.

Documents received

The documents received were as follows:

<i>Document</i>	<i>Version</i>	<i>Date</i>
GP Letter Depression Group	1	05 November 2009
Participant Information Sheet: Non-Clinical Group	2	05 November 2009
Participant Consent Form: Non-Clinical Controls	2	05 November 2009
Participant Information Sheet: Delusion Group	2	05 November 2009
Participant Information Sheet: Depression Group	2	05 November 2009
Participant Consent Form: Patients	2	05 November 2009
GP Letter Delusions Group	1	05 November 2009

You should ensure that the sponsor has a copy of the final documentation for the study. It is the sponsor's responsibility to ensure that the documentation is made available to R&D offices at all participating sites.

09/H0721/61

Please quote this number on all correspondence

Yours sincerely

Ms Kathy Clark
Committee Co-ordinator

E-mail: katherine.clark@royalfree.nhs.uk

Copy to: 8.8.1.1 Jennifer Liebscher, King's College London, R&D Office

Appendix B. Participant information sheets and consent forms

Information sheet for persecutory delusions group

**Institute of
Psychiatry**

**at The
Maudsley**

South London and Maudsley 
NHS Foundation Trust

Department of Psychology
Institute of Psychiatry
Kings College London
De Crespigny Park
London, SE5 8AF

KING'S
College
LONDON
Founded 1829

PARTICIPANT INFORMATION SHEET: PERSECUTION AND DEPRESSION

We would like to invite you to participate in a research study. Before you decide, you need to understand why the research is being done and what it would involve for you. Please tell us if anything is unclear or if you would like more information. Talk to other people about the study if you want to.

Key facts:

- This study is about the fear of other people trying to harm you, and how depression might make this fear worse.
- You would have two meetings with the researcher, six months apart. Each meeting would last about two hours.
- We will ask you about what you usually do to cope with being sad or worried. There will also be some tasks of memory and problem solving.

What is the purpose of the study?

Many people will experience the fear of being hurt by others at some point. But for some people, worries like this can become very strong and long-lasting. We want to find out what makes the fear persist. In a similar way, we all experience sadness sometimes, but some people become depressed for long periods of time. We think that there might be similar reasons for the persistence of sadness and for the persistence of fear. Then similar approaches could be used to help people with these problems.

Different people react differently to feeling worried or sad. For example, some people might spend a lot of time thinking about why they feel upset and what this means. Other people might try to avoid thinking about it at all. Our style of thinking can affect how we remember the past, and our memories of the past affect how we approach the future. Spending too much time focusing on problems might make them more upsetting. On the other hand, if we avoid thinking about our problems altogether then we might not be able to solve them. The right approach to problem solving might help us to feel calmer and happier. We want to look at how people's moods and concerns change over time and how these changes relate to the person's thinking style.

This project is part of a PhD thesis. The outcome of the research will be published in a scientific journal, but individual participants will not be identified. We hope that a better understanding of these processes will help us to improve psychological talking treatments for people who have depression and distressing worries about other people.

Why have I been invited to take part?

120 people will take part in the study. All will be adults (aged 18+) who can speak and read English well enough to complete the assessments. You have been asked to participate because you may sometimes have worries about being harmed by others.

Do I have to take part?

No, you do not have to take part. It is up to you to decide. We will explain the study and go through this information sheet, which is yours to keep. We will then give you a consent form to sign if you agree to take part. You are free to withdraw at any time, without giving a reason. This would not affect your care.

What will happen if I take part?

If you agree to take part, you will have two meetings with the researcher. Each meeting will take approximately two hours. The researcher will ask you about your mood and any worries you might have about being harmed by others. You will also be asked to fill in seven multiple-choice questionnaires. These will ask about what you usually do when you feel sad or worried, for example whether you spend a lot of time analysing the reasons for being upset. Then there will be five verbal tasks which are about memory and problem solving. These are designed to be brief. The second meeting will be six months after the first meeting and the assessments will be the same.

We would like to record your meetings with the researcher using a digital voice recorder. This is so that we can check that the tasks are presented in the same way to all participants. It also makes the meetings shorter because we do not have to write your answers down as you say them. The meetings do not have to be recorded if you do not want them to be.

We would also be very interested to know about your experiences of persecution and depression, and what you thought about taking part in the study. We might want to publish a direct quote from you. Your name would not be published. We will not publish any quotes from you if you do not want us to.

We would like to send a letter to your GP to let him/her know that you are taking part in our research. We will not do this if you do not want us to.

If you agree to take part in the study, we will look at your medical notes to find out about what medication you take and any diagnosis that has been given to you. We will not use your notes for any other purpose.

Expenses and payment

You will be paid £40 in total to cover your expenses and to reimburse you for the time you spend in the study. We will give you £20 after the first meeting and another £20 after the second.

What are the potential risks of taking part?

It is not expected that participation in the study has any risks. However, if you find any of the questions asked upsetting, and would like to talk about this, please tell the researcher.

What will happen to the information I give in the study?

All your answers to the questionnaires and the tasks will be kept on paper and in files on our computer for ten years, and will then be destroyed. Paper questionnaires will be kept in a locked cabinet in a locked office. Computer files containing personal information about you will be encrypted so that nobody except for the researcher can open them. The recordings will be kept as electronic files. They will be kept securely and anonymously and will be identifiable only by a number instead of your name.

The information you give will usually be available only to the research team. However, the researcher will share with your clinical team any important information that is relevant to the care you receive.

In the unlikely event that you become unable to continue to take part in the study, the information that you had given up to that point would continue to be used confidentially for the purposes described. If you requested that your data be withdrawn from the study completely and destroyed, then we would do this.

If you have a concern about the study

If you have a concern about this study, you should speak to us or a member of your clinical team. If you remain unhappy and would like to complain formally, you can do this through the NHS Complaints Procedure. Details can be obtained from the hospital.

All research in the NHS is reviewed by an independent group of people, called a Research Ethics Committee, to protect your safety, rights, wellbeing and dignity. This study has been reviewed and given favourable opinion by the Moorfields and Whittington NHS Research Ethics Committee, reference 09/H0721/61.

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Information sheet for depression group

**Institute of
Psychiatry**

**at The
Maudsley**

South London and Maudsley 
NHS Foundation Trust

Department of Psychology
Institute of Psychiatry
Kings College London
De Crespigny Park
London, SE5 8AF

KING'S
College
LONDON
Founded 1829

PARTICIPANT INFORMATION SHEET: PERSECUTION AND DEPRESSION

We would like to invite you to participate in a research study. Before you decide, you need to understand why the research is being done and what it would involve for you. Please tell us if anything is unclear or if you would like more information. Talk to other people about the study if you want to.

Key facts:

- This study is about how depression might contribute to a fear of being harmed by other people.
- You would meet with the researcher for about two hours.
- We will ask you about what you usually do to cope with being sad or worried. There will also be some tasks of memory and problem solving.

What is the purpose of the study?

Many people will experience the fear of being hurt by others at some point. But for some people, worries like this can become very strong and long-lasting. We want to find out what makes the fear persist. In a similar way, we all experience sadness sometimes, but some people become depressed for long periods of time. We think that there might be similar reasons for the persistence of sadness and for the persistence of fear. Then similar approaches could be used to help people with these problems.

Different people react differently to feeling worried or sad. For example, some people might spend a lot of time thinking about why they feel upset and what this means. Other people might try to avoid thinking about it at all. Our style of thinking can affect how we remember the past, and our memories of the past affect how we approach the future. Spending too much time focusing on problems might make them more upsetting. On the other hand, if we avoid thinking about our problems altogether then we might not be able to solve them. The right approach to problem solving might help us to feel calmer and happier. We want to look at how people's moods and concerns change over time and how these changes relate to the person's thinking style.

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Why have I been invited to take part?

120 people will take part in the study. All will be adults (aged 18+) who can speak and read English well enough to complete the assessments. Some of the participants

in the study have distressing fears about being harmed by other people. You have been asked to participate because you may be experiencing depression.

Do I have to take part?

No, you do not have to take part. It is up to you to decide. We will explain the study and go through this information sheet, which is yours to keep. We will then give you a consent form to sign if you agree to take part. You are free to withdraw at any time, without giving a reason. This would not affect your care.

What will happen if I take part?

If you agree to take part, you will meet with the researcher for around two hours. The researcher will ask you about your mood and any worries you might have about being harmed by others. You will also be asked to fill in seven multiple-choice questionnaires. These will ask about what you usually do when you feel sad or worried, for example whether you spend a lot of time analysing the reasons for being upset. Then there will be five verbal tasks which are about memory and problem solving. These are designed to be brief.

We would like to record your meetings with the researcher using a digital voice recorder. This is so that we can check that the tasks are presented in the same way to all participants. It also makes the meetings shorter because we do not have to write your answers down as you say them. The meetings do not have to be recorded if you do not want them to be.

We would also be very interested to know about your experiences of persecution and depression, and what you thought about taking part in the study. We might want to publish a direct quote from you. Your name would not be published. We will not publish any quotes from you if you do not want us to.

We would like to send a letter to your GP to let him/her know that you are taking part in our research. We will not do this if you do not want us to.

If you agree to take part in the study, we will look at your medical notes to find out about what medication you take and any diagnosis that has been given to you. We will not use your notes for any other purpose.

Expenses and payment

You will be paid £20 to cover your expenses and to reimburse you for the time you spend in the study.

What are the potential risks of taking part?

It is not expected that participation in the study has any risks. However, if you find any of the questions asked upsetting, and would like to talk about this, please tell the researcher.

What will happen to the information I give in the study?

All your answers to the questionnaires and the tasks will be kept on paper and in files on our computer for ten years, and will then be destroyed. Paper questionnaires will be kept in a locked cabinet in a locked office. Computer files containing personal information about you will be encrypted so that nobody except for the researcher can open them. The recordings will be kept as electronic files. They will be kept securely and anonymously and will be identifiable only by a number instead of your name.

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Information sheet for non-clinical group

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Key facts:

- This study is about how depression might contribute to a fear of being harmed by other people.
- You would meet with the researcher for about two hours.
- We will ask you about what you usually do to cope with being sad or worried. There will also be some tasks of memory and problem solving.

What is the purpose of the study?

Many people will experience the fear of being hurt by others at some point. But for some people, worries like this can become very strong and long-lasting. We want to find out what makes the fear persist. In a similar way, we all experience sadness sometimes, but some people become depressed for long periods of time. We think that there might be similar reasons for the persistence of sadness and for the persistence of fear. Then similar approaches could be used to help people with these problems.

Different people react differently to feeling worried or sad. For example, some people might spend a lot of time thinking about why they feel upset and what this means. Other people might try to avoid thinking about it at all. Our style of thinking can affect how we remember the past, and our memories of the past affect how we approach the future. Spending too much time focusing on problems might make them more upsetting. On the other hand, if we avoid thinking about our problems altogether then we might not be able to solve them. The right approach to problem solving might help us to feel calmer and happier. We want to look at how people's moods and concerns change over time and how these changes relate to the person's thinking style.

This project is part of a PhD thesis. The outcome of the research will be published in a scientific journal, but individual participants will not be identified. We hope that a better understanding of these processes will help us to improve psychological talking treatments for people who have depression and distressing worries about other people.

Why have I been invited to take part?

120 people will take part in the study. All will be adults (aged 18+) who can speak and read English well enough to complete the assessments. Some of the participants in the study experience depression and some have distressing fears about being harmed by other people. You have been approached to take part as a non-clinical control participant.

Do I have to take part?

No, you do not have to take part. It is up to you to decide. We will explain the study and go through this information sheet, which is yours to keep. We will then give you a consent form to sign if you agree to take part. You are free to withdraw at any time, without giving a reason. This would not affect your care.

What will happen if I take part?

If you agree to take part, you will meet with the researcher for around two hours. The researcher will ask you about your mood and any worries you might have about being harmed by others. You will also be asked to fill in seven multiple-choice questionnaires. These will ask about what you usually do when you feel sad or worried, for example whether you spend a lot of time analysing the reasons for being upset. Then there will be five verbal tasks which are about memory and problem solving. These are designed to be brief.

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We would also be very interested to know about your experiences of persecution and depression, and what you thought about taking part in the study. We might want to publish a direct quote from you. Your name would not be published. We will not publish any quotes from you if you do not want us to.

Expenses and payment

You will be paid £20 to cover your expenses and to reimburse you for the time you spend in the study.

What are the potential risks of taking part?

It is not expected that participation in the study has any risks. However, if you find any of the questions asked upsetting, and would like to talk about this, please tell the researcher.

What will happen to the information I give in the study?

All your answers to the questionnaires and the tasks will be kept on paper and in files on our computer for ten years, and will then be destroyed. Paper questionnaires will be kept in a locked cabinet in a locked office. Computer files containing personal information about you will be encrypted so that nobody except for the researcher can open them. The recordings will be kept as electronic files. They will be kept securely and anonymously and will be identifiable only by a number instead of your name.

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confidentially for the purposes described. If you requested that your data be withdrawn from the study completely and destroyed, then we would do this.

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Consent form for persecutory delusions group and depression group**CONSENT FORM****Persecution and Depression.**

PLEASE TICK IF YOU AGREE:

- ☐ I confirm that I have read and understand the information sheet. I have had the opportunity to consider the information and to ask questions.
- ☐ I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected.
- ☐ I understand that relevant sections of my medical notes and data collected during the study may be looked at by members of the research team, regulatory authorities, or the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.
- ☐ I understand that my clinical team will be informed of any important medical information that might arise during the study.
- ☐ I agree to take part in the above study.
- ☐ I agree to have my meeting(s) with the researcher recorded with a digital voice recorder.
- ☐ I agree to the researcher publishing direct quotes from me, which will not mention my name.
- ☐ I agree to my GP being informed that I am taking part.

Name of participant-----
Date-----
Signature-----
Name of researcher-----
Date-----
Signature

Consent form for non-clinical group**CONSENT FORM****Persecution and Depression.**

PLEASE TICK IF YOU AGREE:

- ☐ I confirm that I have read and understand the information sheet. I have had the opportunity to consider the information and to ask questions.
- ☐ I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical care or legal rights being affected.
- ☐ I understand that relevant sections of the data collected during the study may be looked at by members of the research team or regulatory authorities, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.
- ☐ I agree to take part in the above study.
- ☐ I agree to have my meeting(s) with the researcher recorded with a digital voice recorder.
- ☐ I agree to the researcher publishing direct quotes from me, which will not mention my name.

Name of participant Date Signature

Name of researcher Date Signature

Appendix C. Study measures

Green et al. Paranoid Thoughts Scale (Green et al., 2008)

Please read each of the statements carefully.

They refer to thoughts and feelings you may have had **about others over the last month**.

Think about the **last month** and indicate the extent of these feelings from **1 (Not at all)** to **5 (Totally)**. Please complete both Part A and Part B.

N.B. Please do not rate items according to any experiences you may have had under the influence of drugs.

Part A.	<i>Not at all</i>		<i>Somewhat</i>		<i>Totally</i>
1. I spent time thinking about friends gossiping about me	1	2	3	4	5
2. I often heard people referring to me	1	2	3	4	5
3. I have been upset by friends and colleagues judging me critically	1	2	3	4	5
4. People definitely laughed at me behind my back	1	2	3	4	5
5. I have been thinking a lot about people avoiding me	1	2	3	4	5
6. People have been dropping hints for me	1	2	3	4	5
7. I believed that certain people were not what they seemed	1	2	3	4	5
8. People talking about me behind my back upset me	1	2	3	4	5
9. I was convinced that people were singling me out	1	2	3	4	5
10. I was certain that people have followed me	1	2	3	4	5
11. Certain people were hostile towards me personally	1	2	3	4	5
12. People have been checking up on me	1	2	3	4	5
13. I was stressed out by people watching me	1	2	3	4	5
14. I was frustrated by people laughing at me	1	2	3	4	5
15. I was worried by people's undue interest in me	1	2	3	4	5
16. It was hard to stop thinking about people talking about me behind my back	1	2	3	4	5

Part B.					
	<i>Not at all</i>	<i>Somewhat</i>	<i>Totally</i>		
1. Certain individuals have had it in for me	1	2	3	4	5
2. I have definitely been persecuted	1	2	3	4	5
3. People have intended me harm	1	2	3	4	5
4. People wanted me to feel threatened, so they stared at me	1	2	3	4	5
5. I was sure certain people did things in order to annoy me	1	2	3	4	5
6. I was convinced there was a conspiracy against me	1	2	3	4	5
7. I was sure someone wanted to hurt me	1	2	3	4	5
8. I was distressed by people wanting to harm me in some way	1	2	3	4	5
9. I was preoccupied with thoughts of people trying to upset me deliberately	1	2	3	4	5
10. I couldn't stop thinking about people wanting to confuse me	1	2	3	4	5
11. I was distressed by being persecuted	1	2	3	4	5
12. I was annoyed because others wanted to deliberately upset me	1	2	3	4	5
13. The thought that people were persecuting me played on my mind	1	2	3	4	5
14. It was difficult to stop thinking about people wanting to make me feel bad	1	2	3	4	5
15. People have been hostile towards me on purpose	1	2	3	4	5
16. I was angry that someone wanted to hurt me	1	2	3	4	5

Beck Depression Inventory II

Image not included for copyright reasons

Image not included for copyright reasons

Beck Anxiety Inventory

Image not included for copyright reasons

PSYRATS: Delusions Scale (Haddock et al., 1999)

Establish central belief during SCAN interview and write down below.

.....

1.	Amount of preoccupation	0	1	2	3	4
2.	Duration of preoccupation	0	1	2	3	4
Avoidance effort				Avoidance success		
3.	Conviction	0	1	2	3	4
4.	Amount of distress	0	1	2	3	4
5.	Intensity of distress	0	1	2	3	4
6.	Disruption	0	1	2	3	4

Questions and Scoring Criteria

1. Amount of preoccupation with delusions

How much time do you spend thinking about?

- all the time / daily / weekly etc.?

- 0 - No delusions, or beliefs which the subject thinks about less than once a week
- 1 - Subject thinks about beliefs at least once a week
- 2 - Subject thinks about beliefs at least once a day
- 3 - Subject thinks about beliefs at least once an hour
- 4 - Subject thinks about delusions continuously or almost continuously. Subject can only think of other things for a few seconds or minutes.

2. Duration of preoccupation with delusions

When the beliefs come to your mind, how long do they persist?

- few seconds / minutes / hours, etc.?

- 0 - No delusions
- 1 - Thoughts about beliefs last for a few seconds, fleeting thoughts
- 2 - Thoughts about beliefs last for several minutes
- 3 - Thoughts about beliefs last for at least 1 hour
- 4 - Thoughts about beliefs usually last for hours at a time

Do you try to avoid thinking about it? How hard do you try (0-100 scale)?

How much do you succeed avoiding it (0 – 100 scale)?

3 Conviction (at the time of interview)

At the present time, how convinced are you that your beliefs are true? Can you estimate this on a scale from 0 – 100, where 100 means that you are totally convinced by your beliefs and 0 means that you are not convinced at all?

- 0 - No conviction at all
- 1 - Very little conviction in reality of beliefs, less than 10%
- 2 - Some doubts relating to conviction in beliefs, between 10 and 49%
- 3 - Conviction in belief is very strong, between 50 and 99%
- 4 - Conviction is 100%

4 Amount of distress

**Do your beliefs cause you distress?
How much of the time do they cause you distress?**

- 0 - Beliefs never cause distress
- 1 - Beliefs cause distress on the minority of occasions
- 2 - Beliefs cause distress on less than 50% of occasions
- 3 - Beliefs cause distress on the majority of occasions when they occur between 50 and 99% of the time
- 4 - Beliefs always cause distress when they occur

5 Intensity of distress

**When your beliefs distress you, how severe does this feel?
How distressing would you say it is on a scale of 0 – 100, where 0 is not at all distressing and 100 is so bad it couldn't be any worse?**

- 0 - No distress
- 1 - Beliefs cause slight distress
- 2 - Beliefs cause moderate distress
- 3 - Beliefs cause marked distress
- 4 - Beliefs cause extreme distress, could not be worse

6 Disruption to life caused by beliefs

How much disruption do your beliefs cause you?

- Do they prevent you working or carrying out a day-time activity?
- Do they interfere with your relationships with family or friends?
- Do they interfere with your ability to look after yourself, e.g. washing, changing clothes, etc.?
- 0 - No disruption to life: able to maintain independent living with no problems in daily living skills. Able to maintain social and family relationships (if present)
- 1 - Beliefs cause minimal amount of disruption to life, e.g. interferes with concentration although able to maintain daytime activity and social and family relationships and be able to maintain independent living without support
- 2 - Beliefs cause moderate amount of disruption to life, causing some disturbance to daytime activity and/or family or social activities. The patient is not in hospital, although may live in supported accommodation or receive additional help with daily living skills
- 3 - Beliefs cause severe disruption to life, so that hospitalisation is usually necessary. The patient is able to maintain some daily activities, self-care and relationships while in hospital. The patient may also be in supported accommodation, but experiencing severe disruption of life in terms of activities, daily living skills and/or relationships
- 4 - Beliefs cause complete disruption of daily life, requiring hospitalization. The patient is unable to maintain any daily activities and social relationships. Self-care is also severely disrupted.

Brief Core Schema Scales (Fowler et al., 2006)

This questionnaire lists beliefs that people can hold about themselves and other people. Please indicate whether you hold each belief by **circling either NO or YES**. If you hold the belief (**YES**), then please indicate how strongly you hold it by **circling a number 1- 4, i.e. you “believe it slightly” (1), you “believe it moderately” (2), you “believe it very much” (3) or you “believe it totally” (4).**

Try to judge the beliefs on how you have generally viewed yourself and others over time. Do not spend too long on each belief. There are no right or wrong answers and the first response to each belief is often the most accurate.

MYSELF			Believe it slightly	Believe it mod.	Believe it very much	Believe it totally
I am unloved	NO	YES →	1	2	3	4
I am worthless	NO	YES →	1	2	3	4
I am weak	NO	YES →	1	2	3	4
I am vulnerable	NO	YES →	1	2	3	4
I am bad	NO	YES →	1	2	3	4
I am a failure	NO	YES →	1	2	3	4
I am respected	NO	YES →	1	2	3	4
I am valuable	NO	YES →	1	2	3	4
I am talented	NO	YES →	1	2	3	4
I am successful	NO	YES →	1	2	3	4
I am good	NO	YES →	1	2	3	4
I am interesting	NO	YES →	1	2	3	4

OTHER PEOPLE			Believe it slightly	Believe it mod.	Believe it very much	Believe it totally
Other people are hostile	NO	YES →	1	2	3	4
Other people are harsh	NO	YES →	1	2	3	4
Other people are unforgiving	NO	YES →	1	2	3	4
Other people are bad	NO	YES →	1	2	3	4
Other people are devious	NO	YES →	1	2	3	4
Other people are nasty	NO	YES →	1	2	3	4
Other people are fair	NO	YES →	1	2	3	4
Other people are good	NO	YES →	1	2	3	4
Other people are trustworthy	NO	YES →	1	2	3	4
Other people are accepting	NO	YES →	1	2	3	4
Other people are supportive	NO	YES →	1	2	3	4
Other people are truthful	NO	YES →	1	2	3	4

Ruminative Response Scale (Treynor et al., 2003)

People think and do many different things when they feel down, sad or depressed. Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each one when you feel down, sad or depressed. Please indicate what you *generally* do, not what you think you should do.

Almost Never	Sometimes	Often	Almost Always	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	1. Think about how alone you feel.
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	2. Think "I won't be able to do my job/work because I feel so bad"
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	3. Think about your feelings of fatigue and achiness
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	4. Think about how hard it is to concentrate
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	5. Think about how passive and unmotivated you feel
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	6. Analyse recent events to try and understand why you are depressed.
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	7. Think about how you don't seem to feel anything anymore
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	8. Think "Why can't I get going?"
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	9. Think "Why do I always react this way?"
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	10. Go away by yourself and think about why you feel this way
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	11. Write down what you are thinking about and analyse it
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	12. Think about a recent situation, wishing it would have gone better
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	13. Think "Why do I have problems other people don't have?"
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	14. Think about how sad you feel

Almost Never	Sometimes	Often	Almost Always	
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	15. Think about all your shortcomings, failings, faults and mistakes
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	16. Think about how you don't feel up to doing anything
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	17. Analyse your personality to try and understand why you are depressed
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	18. Go someplace alone to think about your feelings
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	19. Think about how angry you are with yourself
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	20. Listen to sad music
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	21. Isolate yourself and think about the reasons why you feel sad
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	22. Try to understand yourself by focusing on your depressed mood
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	23. Think "What am I doing to deserve this?"
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	24. Think "I won't be able to concentrate if I keep feeling this way".
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	25. Think "Why can't I handle things better?"

Penn State Worry Questionnaire (Meyer et al., 1990)

Please read each statement and circle an appropriate number: 1, 2, 3, 4, or 5, which indicates how much the statement applies to you.

1 = not at all typical of me, 5 = very typical of me.

	Not at all typical of me			Very typical of me	
1. If I do not have enough time to do everything, I do not worry about it	1	2	3	4	5
2. My worries overwhelm me	1	2	3	4	5
3. I do not tend to worry about things	1	2	3	4	5
4. Many situations make me worry	1	2	3	4	5
5. I know I should not worry about things, but I just cannot help it	1	2	3	4	5
6. When I am under pressure I worry a lot	1	2	3	4	5
7. I am always worrying about something	1	2	3	4	5
8. I find it easy to dismiss worrisome thoughts	1	2	3	4	5
9. As soon as I finish one task, I start to worry about everything else I have to do	1	2	3	4	5
10. I never worry about anything	1	2	3	4	5
11. When there is nothing more I can do about a concern, I do not worry about it any more	1	2	3	4	5
12. I have been a worrier all my life	1	2	3	4	5
13. I notice that I have been worrying about things	1	2	3	4	5
14. Once I start worrying, I cannot stop	1	2	3	4	5
15. I worry all the time	1	2	3	4	5
16. I worry about projects until they are all done	1	2	3	4	5

Acceptance and Action Questionnaire (Hayes et al., 2004)

Below you will find a list of statements. Please rate the truth of each statement as it applies to you. Use the following scale to make your choice.

	1	2	3	4	5	6	7
	Never True	Very Rarely True	Seldom True	Sometimes True	Frequently True	Almost Always True	Always True
1. I am able to take action on a problem even if I am uncertain what is the right thing to do.	1	2	3	4	5	6	7
2. I often catch myself daydreaming about things I've done and what I would do differently next time.	1	2	3	4	5	6	7
3. When I feel depressed or anxious, I am unable to take care of my responsibilities.	1	2	3	4	5	6	7
4. I rarely worry about getting my anxieties, worries, and feelings under control.	1	2	3	4	5	6	7
5. I'm not afraid of my feelings.	1	2	3	4	5	6	7
6. When I evaluate something negatively, I usually recognize that this is just a reaction, not an objective fact.	1	2	3	4	5	6	7
7. When I compare myself to other people, it seems that most of them are handling their lives better than I do.	1	2	3	4	5	6	7
8. Anxiety is bad.	1	2	3	4	5	6	7
9. If I could magically remove all the painful experiences I've had in my life, I would do so.	1	2	3	4	5	6	7

Autobiographical Memory Test (Williams & Broadbent, 1986)

I am interested in your memory for events that have happened in your life. I am going to read to you some words. For each word I want you to think of an event that happened to you which the word reminds you of. The event could have happened recently (yesterday, last week) or a long time ago. It might be an important event, or trivial event.

*Just one more thing : the memory you recall should be a specific event – **an event that lasted less than a day, and occurred at a particular time and place.** So if I said the word “good” – it would not be OK to say, “I always enjoy a good party”, because that does not mention a specific event. But it would be OK to say “I had a good time at Jane’s party” (because that is a specific event). It is important to try retrieve a different memory or event for each cue word. Let us try some words for practice:*

**Car
Tree
Chair**

60 SECONDS PER ITEM

Prompt: “Can you think of a specific time? One particular episode”

Cue Words

Form A was given in the first assessment session, and Form B was given at follow-up to participants with delusions, to avoid practice effects. Positive and negative words were alternated, with a negative word first and a positive word last.

Form A		Form B	
Positive	Negative	Positive	Negative
friendly	tired	happy	failure
pleasant	weakness	excited	hurt
proud	guilty	relieved	bored
lucky	ugly	peaceful	hopeless
carefree	sad	sunny	rejected
calm	mistake	hopeful	blame

Means-Ends Problem Solving Task (Platt & Spivack, 1975)

Instructions

"In this procedure we are interested in how you solve problems. You will be given a number of stories to complete. For each story you will be given the beginning of the story and how the story ends. We'd like you to provide the ideal strategy that will allow the beginning and end of the story to become connected. We would like you to describe this strategy in very specific terms so that it would be possible for anyone to follow your plan of action."

Situations (male form for male participants)

1. H. loved his girlfriend very much, but they had many arguments. One day she left him. H. wanted things to be better. The story ends with everything fine between him and his girlfriend. You begin the story with his girlfriend leaving him after an argument.
2. Mr. C. had just moved in that day and didn't know anyone. Mr. C. wanted to have friends in the neighbourhood. The story ends with Mr. C. having many good friends and feeling at home in the neighbourhood. You begin the story with Mr. C. in his room immediately after arriving in the neighbourhood.
3. Joe is having trouble getting along with the foreman on his job. Joe is very unhappy about this. The story ends with Joe's foreman liking him. You begin the story where Joe isn't getting along with his foreman.

The female form (for female participants) is identical except for the sex of the protagonist and the love interest.

Alternate form for re-assessment

4. One day Al saw a beautiful girl he had never seen before while eating in a restaurant. He was immediately attracted to her. The story ends when they get married. You begin when Al first notices the girl in the restaurant.
5. John noticed that his friends seemed to be avoiding him. John wanted to have friends and be liked. The story ends when John's friends like him again. You begin where he first notices his friends avoiding him.
6. Mr. P. came home after shopping and found that he had lost his watch. He was very upset about it. The story ends with Mr. P. finding his watch and feeling good about it. You begin the story where Mr. P. found that he had lost his watch.

Beads Task (Garety et al., 1991)

Here we have 2 jars: a mainly red jar containing 60 red beads and 40 white beads, and a mainly white jar containing 60 white beads and 40 red beads.

As you can see, the beads have been mixed up in the jars.

I am going to choose one of the jars at random. Beads will be drawn from the selected jar and shown to you one by one. The beads will always come from the same jar and they will be replaced afterwards so that the proportions stay the same.

It is your job to decide which jar the beads have come from, the Mainly White Jar or the Mainly Red Jar. There is a diagram here to remind you of what they look like. You may see as many beads as you like before making a decision. After a bead has been shown to you, you can either ask to see another bead or you can tell me that you know which jar has been chosen, and you can tell me whether it is the Mainly Red Jar or the Mainly White Jar.

Remember, you can see as many beads as you like before you decide which jar the beads are from. Only decide when you are certain.

You will now see the first bead.

White

It is a White bead.

Do you want to see another bead or have you decided?

Red

Red

White

White

Red

White

White

White

Red

White

White

White

White

Red

Red

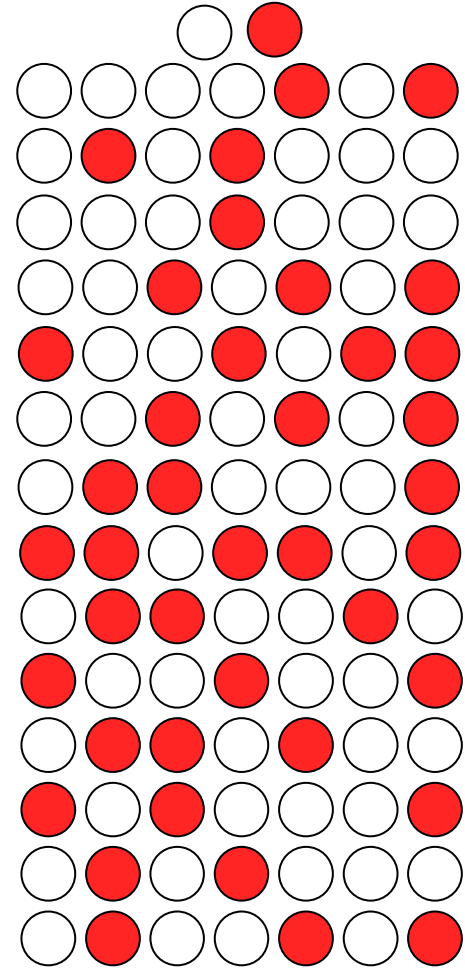
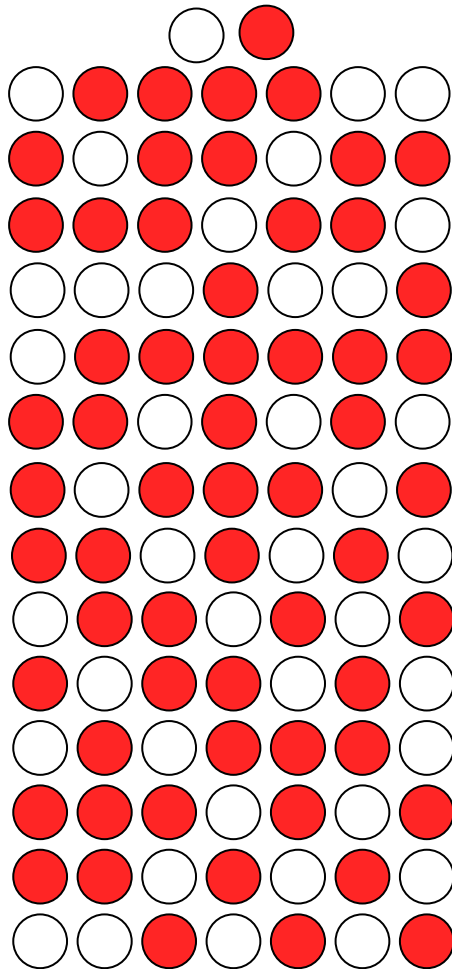
White

Red

Red

White

You must decide now

Beads Task illustrated record sheet for participants

Beads you have previously seen:

Wechsler Test of Adult Reading (Ginsberg, 2003)

Image not included for copyright reasons

FAS verbal fluency test (Benton & Hamsher, 1976)

I'm going to say a letter of the alphabet and I want you to give me as many words that begin with that letter as quickly as you can. For example if I say 'B' you might give me bad, bed.

There are only two rules. I don't want you to give me words that are proper names or places, such as 'Bob' or 'Blackpool'. Also, do not use the same word again with a different ending such as 'eat, eating, eaten,' as that would be cheating.

Begin when I say the letter.

The letter is **S**.

Alternate version for re-assessment:

The letter is **A**.

***Appendix D. Delusional beliefs of the participants with
persecutory delusions***

Persecutory delusions without depression: PD group

The Greek gang are messing with my mind to make me a drug dealer and a prostitute.

The voices are trying to upset me and get me in trouble.

They are putting me to pain to punish me and make me commit suicide.

They are trying to upset me, confuse me, make it look like I'm having a breakdown.

The people from earlier in life intend to kill me.

If I go out, someone will attack me.

My boyfriend and my family are trying to upset me so they can control me.

The people in the street will try to kill me (all of them).

People in the street react to me aggressively and it intimidates me.

Danny Eichman Cohen & Noel are coming in at night and injecting me with HIV blood, and kicking me in the back.

The Turkish community are gossiping about me and being disrespectful.

The devil and the hospital people are trying to kill me.

The police are hassling me, trying to exploit me and destroy me.

My neighbour is harassing me with noise to upset me and provoke me.

Mark & his friends are persecuting me, trying to steal my ID and rob me.

Certain members of society are impeding my progress, possibly through confusion and sabotage.

The African people are persecuting me to take advantage of me for money.

T.O.R. is persecuting me to try to make my life a misery.

The bad spirits are persecuting me to upset me.

My family are stealing my identity to take my wealth.

The people in the street are persecuting me, and I don't know whether it's because of iniquity.

My neighbour is persecuting me to make me move out.

The devil is using people to interfere with my objectives.

Colleagues are talking about me negatively to undermine me.

The Hindus are torturing me with the voices.

People will make false allegations against me to get me in trouble.

Nicoletta is persecuting me with police surveillance.

Certain people want to upset me to bring me down.

The devil is persecuting me with voices.

John is persecuting me with surveillance and bringing me down.

Persecutory delusions with depression: PD+D group

Nigel is trying to make my life so unbearable that I have to move out and become homeless.

PC is hurting me (not physically, but mentally), stressing me out and making me angry.

The neighbours are poisoning me, breaking stuff, taking my things, trying to make me go crazy.

The spirits are trying to make me commit crimes and lead a bad life.

My mum is trying to ruin my life and bring me down.

They [old neighbours] are persecuting me and trying to make me kill myself.

They are going to kill me.

They are trying to kill me and threaten me with black magic.

The people are interfering with my mind and trying to hurt me.

If I go back to the flat, someone will come and kill me.

The voices are hurting me physically and trying to get me in trouble.

Bandek is torturing me, hurting my body.

My two neighbours are trying to make my life a misery.

Mr N and his gang will come and kill me.

The funky people and the regular people are plotting to kill me.

My brother is persecuting me and trying to kill me.

There is a conspiracy to make me have another breakdown.

The people in the street are singling me out to hurt me, physically and mentally.

People will stop me achieving what I want in life.

My neighbours are trying to get rid of me and they might attack me.

The gang are attacking me somatically and psychosomatically.

The men in the BT vans will hurt me.

The gunman will come into my house at night and kill me.

The voices are trying to upset me to bring me down.

The people in the street will attack me.

People in the street will attack me.

My neighbours are trying to piss me off, to get me in trouble.

The people from the ward would hurt me if they could.

The people in the street will attack me.